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Table of Contents.

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ORIGINAL ARTICLES— Page.

On the Management of the Juvenile Sex Offender, by H. M. North, M.B., D.P.M. 685
The Psychiatric Sequelæ of Head Injuries, by B. H. Peterson, M.C., D.P.M. 689
Ten Cases of Rheumatoid Arthritis Treated with PAS and other Measures over a Two-Year Period, by Brian Haynes 692
The Heaf Multiple Puncture Test Compared with the Mantoux Test in Epidemiological Surveys, by E. M. Rathus, M.B., Ch.B. 696
Molluscum Sebaceum, Self-Healing Epithelioma and Tumour-Like Keratosis, with Report of a Case, by Basil A. Stoll, F.F.R., M.R.C.S., D.M.R.T. & D. 698
Staphylococcal Pneumonia, by John Sullivan 700
The Spot-Map in Epidemiology: A Note on the Use of Spot-Maps during Two Epidemics of Poliomyelitis in Western Australia, by D. J. R. Snow 702

REPORTS OF CASES—

Sporotrichosis: A Case Report from Queensland, by C. C. J. Minty, D.T.R., M.C.R.A., M. Mead and M. F. McCaffrey 704
An Adamantinoma in a Dentigerous Cyst, by Richard Flynn and R. Tinning 705
Full-Term Pregnancy and Confinement of a Primipara with Triplets, by P. W. Gill and J. S. Jonas 707
White Sponge Nævus of the Mucosa (Cannon), Nævus Spongiosus Albus Mucosæ: Report of a Case, by L. G. Abbott and L. A. Musso 708

REVIEWS—

Genital Prolapse and Allied Conditions 709
Hypnotic Suggestion: Its Role in Psychoneurotic and Psychosomatic Disorders 710

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—

The London Medical Handbook 710
The Work of the Mental Nurse 710
Aviation Medicine 710
Synopsis of Audiology 710

BOOKS RECEIVED 710

LEADING ARTICLES—

Report on the Health of Great Britain 711

CURRENT COMMENT— Page.

The Evaluation of Hypotensive Drugs 713
Leuchæmia Research 714
Heredity and Glaucoma 715

ABSTRACTS FROM MEDICAL LITERATURE—

Medicine 716

CLINICO-PATHOLOGICAL CONFERENCES—

A Conference at Sydney Hospital 718

OUT OF THE PAST 722

CORRESPONDENCE—

F.O.C.L.A. Post-Graduate Week 722
Toxicity of "Xylocaine" 722
Coronary Disease and Myocardial Infarction 722
Surgery of Hand Trauma 723
The Treatment of Ulcerative Colitis 723

POST-GRADUATE WORK—

The Melbourne Medical Post-Graduate Committee 724
F.O.C.L.A. Post-Graduate Week 724

PUBLIC HEALTH—

Police Offences (Amendment) Act, 1908, as Amended, of New South Wales 725

OBITUARY—

Eulalia Richards 725
John Joseph Witton Flynn 725

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—

New South Wales 726

THE ROYAL AUSTRALASIAN COLLEGE OF PHYSICIANS—

Annual Meeting 726

NAVAL, MILITARY AND AIR FORCE—

Honours and Awards 727
Appointments 727

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 727

NOTICE—

Post-Graduate Golf Match 728
Special Group on Aviation Medicine (British Medical Association) 728

NOMINATIONS AND ELECTIONS 728

DIARY FOR THE MONTH 728

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 728

EDITORIAL NOTICES 728

ON THE MANAGEMENT OF THE JUVENILE SEX OFFENDER.[1]

By H. M. NORTH, M.B., D.P.M.,
Sydney.

THE literature on the subject of the sex offender is notoriously unsatisfactory and shows little cohesion or consistency. Undoubtedly the most important work on sex is Freud's classic "Three Contributions to the Theory of Sex". My copy is a translation of the second reprinting of the second edition. The author's preface to the second edition is dated at Vienna, December, 1909. The date of the first edition is not shown. In spite of the floods of literature on sexual perversions which have since been poured out, this work has not yet been superseded and remains quite up to date.

I am not concerned with adult crime or anomaly and indeed have had little experience of this interesting subject. But I have been furnishing reports on juvenile offenders for our children's courts since 1936.

With his love of the figure of speech known as oxymoron or contradiction in terms, Freud has asserted that every normal child is "polymorphous perverse". What he means, of course, is that what would be regarded as perversion in an adult may be quite normal in a child or juvenile. On this reasoning, I suppose, we may regard the adult pervert as just an individual who has failed to grow up in this particular area, and in reporting on juveniles I have always sheltered behind the concept of "polymorphous perverse" and asserted that the lad has still time to grow up. For instance, if one is asked to express an opinion as to whether Tommy Jones is a homosexual, one may recall Havelock Ellis's dictum (1933) that homosexuality should never be diagnosed before the age of twenty-five years.

If one desires to avoid the bizarre jargon of the psychoanalysts, there is an excellent chapter devoted to juveniles, which, of course, leans heavily on "the Three Contributions" of Freud, in Havelock Ellis's "Psychology of Sex" (1933).

Doshay (1943) makes a useful distinction between the mixed and the primary offender. In his conception, in the mixed offender the sex offence is but an episode in an anti-social career, in which many offences not of a sexual nature also figure. He accepts a target of self-indulgence and pays little heed to the welfare or feelings of his fellows. On the other hand, the primary offender is guilty of the isolated sex offence or offences, but is otherwise a self-respecting individual, who has shown promise of developing into a useful citizen. In Doshay's concluding chapter he makes the following remarks:

[1] The material presented in this paper was obtained during the writer's term of office as Assistant Director and Senior Psychiatrist, School Medical Service, New South Wales.

> Never yet has a boy appeared in the clinic who sought to justify a sexual offence, however insignificant. . . . Even among the aggressive membership of the mixed group, where the moral cultural pattern is to some extent nullified by the very low ethical standards and general personality disorganization, only eight sexual failures appear among the original 148 juvenile sex cases.

Scope of the Present Study.

The present study is based on an analysis of 51 cases referred to the Yasmar Child Guidance Clinic by various children's courts between June, 1953, and September, 1954. These are summarized in Table I. When a boy was granted probation at the clinic's recommendation, in no case is he known to have fallen into the hands of the police again through a further sex offence.

This is a far better result than can be claimed in any other class of offence. Certainly the follow-up is not to be compared in thoroughness with Doshay's. But experience shows that when a primary offender does not return within a few months he gives no further trouble. Regarded from the lowest point of view, he has at least learned to take precautions against being detected again.

In accordance with Doshay's classification, 27 are primary offenders and 23 belong to the mixed group, while one patient (Case I) was suffering from schizophrenia.

Court Appearance.

I regard the court appearance as of prime importance for a favourable outcome. It would be my advice to child guidance clinics, when there has been any degree of aggressiveness or violence, to insist on the child's being charged and made to appear before court as a condition of subsequent acceptance by the clinic.

The court appearance inevitably involves the whole family much more effectively than the unaided clinic can ever manage alone. When the parents take their seats behind the offender in court they get for the first time a queer feeling that they are on trial too. This arouses defensive and protective attitudes towards the offender, though on the way to court they may have regarded him as a monster "ripped untimely from his mother's womb".

There is usually a remand for a short period pending the furnishing of a report by the clinic.

The atmosphere is now optimal from the point of view of the clinic, which is seen as the last shield for both offender and parents from the full rigor of the law. Inexperienced presiding magistrates at times feel that details of the offence should be kept from mothers, and may refuse to admit them to court. Mothers have complained to the social worker that they do not know what it is all about and that they can get no information from anyone but are hedged about by a cloak of silence which they cannot penetrate. This is a big mistake. However nauseous the evidence, the mother's attendance at all court sittings should be required.

Sex Instruction.

For sex instruction I usually arrange a group of four boys who may not necessarily all be before the court for a sex offence. At once the offender feels that he is not the only one in trouble, and he is more likely to talk freely after he has heard the others mention their difficulties. It should be realized that these boys do not need a scientific exposition of anatomy and physiology so much as a workable and working philosophy of life.

We start with David Farrell's little book "Sex Education in Pictures". The high artistic quality and the chaste presentation of the plates always produce an atmosphere of sobriety, while the story of the author's crippling, which did not prevent his high achievement as anatomical draughtsman to the Medical School of the University of Sydney, affords emotional release.

One then proceeds to a general discussion of the psychology of sex by pointing out that while in many respects the sexual impulse in man and animals runs parallel, there are many important differences. Since the human young need two parents for many years, human nature must provide for this or the race would die out. Human nature is such that full sexual satisfaction can be achieved only by taking one mate for life and by cherishing and protecting her and her young.

Here the boys are each in turn invited to express an opinion as to how long they feel they will need the care of both mother and father. This may be a harrowing procedure when one of the class has been deprived of his father's care by bereavement, desertion or inadequacy. The instructor is at no pains to spare him. The sympathetic emotions of the group are stirred and we may achieve their purification in some degree as aimed at by the Greek tragedians. Boys with good fathers pray that they may never be guilty of bringing such sorrow on anyone belonging to them.

Brief discussion of the different patterns of sex behaviour in non-human species is undertaken to accentuate the differences peculiar to human beings. Attention is drawn to the relative submergence of sexual desire in the general patterning of home building and caring for young, as marriage progresses beyond its first exciting weeks. If spouses must continue intercourse every night, we are safe in assuming that they are not happily married. They have married for some reason other than true love and are trying to satisfy themselves on a subhuman level; but this only leaves them desirous.

Persons who have failed to achieve mature sexual satisfaction may seek to find gratification in diverse other ways — for instance, by seducing young girls or by indulging in homosexuality, if they have failed to realize that happiness can be attained only by self-denial and by trying to "give" to others.

Some explanation of the law regarding the age of consent and the liability of the father for the support of illegitimate children may be introduced.

To convey the deep necessity of sacrificing one's own impulses for the sake of another's happiness, as the only way to self-fulfilment, a few biblical or other well-known stories of famous characters may be related by members of the group if possible.

This should give the group the opportunity to enter into more general discussion of any aspects of the subject. One avoids all "must nots", bearing in mind that sex is one of Nature's imperatives. There is emphasis on the fact that the offender against property offends against the laws of the land; but the sex offender is offending against himself, against what is most sacred and most intimate in his personality. It is usually not difficult to make him realize this.

Masturbation.

Physiological explanation of masturbation is given with the aid of the plates in Farrell's book, and the boys are reassured about the dire consequences which uninformed opinion still attaches to it. They are warned that boys who masturbate too freely are in danger of being led on to seek gratification in some other way and may get themselves into serious trouble. Therefore they are advised to make a strict rule not to masturbate more often than once per fortnight and to occupy themselves with sport or study or with helping others instead of having to resort to this last escape of the bored.

The Nature of the Offence.

Table I gives a statistical account of the offences committed. Only the following of these categories call for special discussion.

Indecent Exposure.

Indecent exposure is one of the commonest offences, and at least in the initial stages it responds well to treatment provided the subject is not incarcerated. It is a social malady, and nothing is gained by separating the patient from society. It is not usually a case of exhibitionism in the common acceptance of the term, though it may become

so if not skilfully handled. It is a blind and crude invitation to cohabitation, which can never succeed. The exposure may be accompanied by attempts at lascivious handling or interference with the clothing of the female. Such grabbing may also occur without exposure. Masturbation and the accompanying fantasies are often factors. Pornographic literature, lewd conversations and other forms of sexual excitement may be precursors.

Offences against Small Girls and Boys.

The group who commit offences against small girls and boys is composed of individuals of very immature character, who would never dare to approach a female of contemporary age. Some will give an account of sexual conquests and prowess which are entirely fictitious. Indeed, if a girl of his own age was to make advances to such an offender, his response would be one of acute alarm and escape reaction.

TABLE I.

Offence.	Number of Cases.
Attempted rape	3
Carnal knowledge	2
Carnal knowledge in company	9
Assault on adolescent girl	4
Indecent assault on adult female	1
Indecent assault on small girl	9
Indecent assault on small boy	3
Indecent behaviour	2
Indecent exposure	6
Sex play with adolescent males	33
Homosexual relations with adult	9
Total	51

Homosexual Activities.

The best boarding-schools are not unfamiliar with homosexual practices, and by and large they are well dealt with by the masters. In my experience they are never precursors of adult homosexuality, and unless badly handled leave no after-effects.

A peculiar manifestation of the homosexual stage of adolescent development often appears in the later school years or post-school years. A group of a dozen or even a score of youths may be associated in lewd conversation. Someone procures the services of a depraved female, and one after another they attempt coitus with her. When such an offender is examined it is soon obvious that all the stimulation has come from his male companions. He has only used the female for the purpose of mutual "*masturbatio intra vaginam*". He has noticed nothing whatever about her. He does not know her name and cannot tell any of her personal characteristics. Of the group of offenders in the present study, 18% fell in this category.

More serious forms of homosexuality always date back to fixations in early infancy and are usually associated with an unresolved Œdipus complex. Even though such youths may not be brought before the court again, one always feels grave doubts about the ultimate outcome. Many instances are to be found in psychoanalytical literature. Here it is proposed to call attention to two mechanisms, which one meets again and again.

Mother Fixation.—For some reason the mother insists on bringing up her boy as a girl. It may be that she herself is sexually immature and cannot adjust herself to her marriage. She has much too heavy an emotional lean on her boy, who is educated in all the female domestic arts and crafts; but sport or anything else which may bring him into association with other boys cannot be countenanced. He grows up incapable of filling the masculine role. All heterosexual activity is equated with his relationship to his mother and is blocked by the incest taboo.

Paternal Deprivation.—The mother may have a strong personality and be well adjusted. She may be a widow, a divorcee or a deserted wife, or the father may be inadequate and held in contempt by the mother and children. He may be an absent-minded professor who has long since forgotten that he has ever been blessed with a son, except when he has to meet bills. The boy is very hungry for a father's care. Why does not his father treat him as other boys' fathers do? One day this boy will be loitering on a railway station or wistfully scanning the posters outside a picture theatre when he is accosted by a well-dressed and seemingly respectable gentleman, who tells him of a very good picture he would like him to see. However little this youth has figured in scientific literature, his psychology is well understood by a certain section of adult perverts in the community. Weeks and months may pass before any sexual advance is made. The interval is filled with music, art, theatres and all the things the boy feels he has missed with his own father. At length the day will come, while the boy is absorbed in a thrilling picture. The patron's hand will fall in the genital area as if by accident. The boy is embarrassed but does not protest. He is already accustomed to quite paternal caresses. I cite a case.

CASE I.—R. was an adopted child with five adoptive sisters. His adoptive mother had always wanted a son, but could produce only daughters. R. was not very old when she died. The father and sisters had never wanted R., but had given him every material care for the dead mother's sake. They denied him nothing, but there was no warmth in the relationship. At the age of fourteen years R. was accosted outside a picture theatre. His new patron showed him all the warmth he had so much missed in his own home and it was many weeks before seduction was finally accomplished.

R. was a bright lad of artistic temperament. The Vocational Guidance Bureau advised signwriting as a trade and found him an apprenticeship. Through several of the war years R. came to me at monthly intervals. I tried to make him interested in sport and various social activities. He would listen to me out of courtesy, but as soon as an opening occurred he would start to tell me with a great deal of dramatization of the last film he had seen. It would now be my turn to have the courtesy to listen to his excited discourse. In pauses I would hint that he would be late for his technical college class. R. was working very hard, and it was usually about 5.30 p.m. when he called; I was lucky if I managed to dismiss him by 6.30 p.m. I would suggest that therapy might soon be terminated, but he would insist on another appointment. Finally a night came when I said: "I won't make another appointment, but you may pop in any time you like and I shall be pleased to see you."

The next morning R. was arrested in a residential at Wollongong, where he had spent the night with an adult pervert picked up on Wynyard Station.

Only then did it occur to me that over all these months R. had quite unconsciously been trying to woo me just as his first patron had wooed him, by means of film dramas. As long as he was sure of his one hour per month with me he was satisfied. A physical relationship was not necessary to him, but the threat of my withdrawal was too much for him and he could see no prospect of forming another similar relationship except on a physical basis.

There are two other groups of homosexuals which require mention.

The Juvenile Male Prostitute.—The motivation of the male prostitute is not very different from that of the female prostitute. It is an easy and lucrative livelihood for an idle and luxurious individual. Though there is no example in the present series, it is by no means a rare problem.

The Luxurious Homosexual.—The luxurious homosexual figures principally in adult delinquency. His difficulty is that he has never learned to make any self-sacrifice, and mature sexual activity inevitably involves sacrifice. Therefore he seeks to buy sexual accommodation. If he uses a female there is always the risk of pregnancy and of the responsibilities of paternity. With a male partner this risk disappears. I cite a case, in which there was certainly evidence that the Œdipus complex had not been satisfactorily resolved. Though a borderline case, it may be best considered in this group.

CASE II.—E. had served several reformatory terms, not all for sexual offences, before he was referred to a child guidance clinic, and would reach the age limit for the jurisdiction of the children's court in a few months. When arrested for his present offence he was very attractively dressed as a female, and the fact that he had the misfortune to accost a plainclothes detective may throw some doubt on a commonly held theory, that homosexuals have an uncanny knack of recognizing their own sort.

During the war years E. called on me on several occasions, though no systematic therapy was ever attempted. For instance, he came about his exemption from military service, alleging that it was torture to him to have to sleep in barracks with a lot of other men.

One day he burst into my room in great excitement. He stated that he had just read in a magazine how medical science had now discovered how to change one's sex surgically. He demanded that I arrange an operation forthwith, alleging that he had all the instincts of a woman. I dissented and hinted that he was only self-indulgent. As our argument proceeded, I eventually conceded that I would believe that E. had some of the instincts of a woman if he would take a new-born baby, feed it, wash it and care for it in every respect throughout the twenty-four hours of every day for six months. E. became quite deflated. He had no reply to this.

Algolagnia.—Sexual activity associated with the infliction of suffering is rarely a persistent problem in juveniles. I do not propose to enter into any discussion whether the enjoyment of cruelty by small children should be regarded as a sexual activity. Since the Freudians hold that all behaviour is derived ultimately from the sexual impulse, they have no alternative to seeking a sexual explanation. The reader may be referred to psychoanalytical literature for the mechanisms of origin of sadism and masochism. However, many cases may be explained, without the need to delve into deeply unconscious processes, by a mechanism similar to that described in the case of the luxurious homosexual. Indeed, they often all occur in the same individual. The algolagnist is incapable of romantic love and attempts to satisfy his sex on an animal level. But he experiences no satisfaction, and in his frustration he attacks his partner. The masochist is similarly motivated, but instead of satisfaction he experiences only disgust and humiliation from his beastly performance. He seeks relief in punishment.

On the Management of the Individual Offender.

At a time when the Kinsey reports are very much before the public, the question arises as to whether we may endeavour to inculcate ideals of chastity or whether we should be satisfied with instructing our patients how to avoid falling into the hands of the police again in the future.

In dealing with the primary offender one often feels that the trouble is that he has been too strait-laced and finds it the irony of fate that he should be detained in the shelter while so many of his loose-living acquaintances are at large, free to pursue their nefarious practices. He has denied the existence of his sexual impulses instead of coming to terms with them. In an unguarded moment his sex has asserted itself in the crudest manner. The problem is to promote a derepression and to introduce him to socially acceptable forms of sexual expression. The most distinctive events in human sexuality have no counterpart in animal sexuality. There is nothing to prevent a celibate participating in a fully socially acceptable manner. The subject needs to be coached in the relative unimportance of the act, which an idiot can perform just as well as he can. But it is well to remind him that even in marriage it is not wise to perform like an idiot or beast. W. Shakespeare's prescription seems appropriate, "got 'tween asleep and wake", unless in this epoch of ultra-scientific advancement we prefer artificial insemination or a test-tube baby.

The secondary offender, on the other hand, voluntarily sets out to indulge himself. We aim to show him the satisfaction his ancestors all down the ages have derived from making sacrifices. We are sure that sacrifice is a primal human need, just as much as sex. Nowadays we do not make sacrifice by libation or burned offering, but we make sacrifice just as effectively by forgoing our impulses. We cannot be happy otherwise.

In the case of neither the primary nor the secondary offender do we discuss the ideal of chastity, except to hint that it has everything to recommend it from the point of view of health both of body and of mind.

Court chaplains feel that they have a special function to fulfil with sexual offenders, and the court will often make it a condition of probation that the lad attend the chaplain for religious instruction. We heartily endorse this practice.

Whether one is a theist or an atheist, one cannot deny that one's ancestors over thousands of years have been deeply concerned with religion and have derived profoundest satisfaction from making sweet sacrifice to the Lord their God. Whether one regards religion as a response to the prime mover, the Creator of heaven and of earth, or as having arisen as a eugenic variation which has contributed to the survival of the species, or with the Lamarckians attribute religion to the transmission of acquired characters, there can be no doubt that theist and atheist alike are endowed with anatomical neural centres devoted to religion.

Kinsey has made a profound study of sexual practices in the United States of America. Conditions may or may not be very similar here (Editorial, 1954). We do not know, until a similar research has been undertaken among our own people. What Kinsey has proved is that sexual morality is certainly very loose in the United States. The biblical chronicler also found sexual licence rife in the ancient cities of Sodom and Gomorrah. He interpreted his findings in a way very different from Kinsey. In any case, satisfactory standards of sexual hygiene will never be determined by statistical methods. If I can point to a rare clean-living man and to a rare clean-living woman, who enjoy both good health and happiness, I have proved that the ideal of chastity is not unattainable.

If a community permits a high degree of sexual licence, should we not regard it as a foolish indulgence, tending to bring about personal unhappiness and social disintegration?

For McDougall, the crude animal appetite and a high-minded sentiment of love have little in common. The latter he would equate with other sentiments, such as love of country, artistic and scientific devotion, and even abstract sentiments such as truthfulness, loyalty, honesty and faithfulness.

All down the ages the theme of love has inspired great literature and art. Seer and prophet, poet and philosopher have alike made it their inspiration. There is a danger that, when the medical scientist, biologist or taxonomist undertakes to impart sexual instruction he may be too obsessed with the pathological. We want to show our instructee how good men can be rather than how bad.

Acknowledgements.

I am obliged to Dr. H. G. Wallace, Director-General of Public Health, for permission to refer to cases from the Department's files. I also owe my thanks to the late Miss Victoria Thorpe, B.A., for psychological examinations, including character ratings, and to Miss Faith Alleyne, B.A., Dip.Ed., for family studies.

Mr. Frank Murphy, presiding magistrate of Yasmar Children's Court, has read my manuscript and offered me valuable encouragement. I have to express a special debt of gratitude to Miss Beryl Wright, B.Ec., of Hong-Kong University, who has collaborated with me over many years in the study of juvenile delinquents, for reading my manuscript and for making many valuable suggestions.

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THE PSYCHIATRIC SEQUELÆ OF HEAD INJURIES.[1]

By B. H. Peterson, M.C., D.P.M.,
Sydney.

I should like to begin with a quotation from Richardson (1951): "The difficult subjects of headaches, dizziness and mental changes after head injury have been written about extensively, though inconclusively." However, a brief survey of the present state of our knowledge may help to organize our thinking and indicate the main problems. One problem, for instance, concerns the relative importance of organic brain damage and of psychogenic influences in the production of psychiatric illness after head injury. In this regard the conclusions of Goldstein (1952) are interesting. He considers that it is a fault in principle to try to distinguish between the so-called "organic" and "functional" illnesses as far as symptomatology and therapy are concerned. In both conditions we are dealing with an abnormal functioning of the same psycho-physical apparatus, and with the attempts of the organism to adjust itself to this. It is our task to help the patient in this adjustment by every known physical and psychological means.

The distribution of these cases (mostly closed head injuries) in our present psychiatric set-up calls for comment. The mental hospital psychiatrist sees very few of them. When he does he is so thrilled that he often shows the patient at a clinical meeting. During the year ending June 30, 1953, there were 2006 patients admitted for the first time to mental hospitals in New South Wales. In eight of these cases a previous head injury was regarded as the main ætiological factor. This is an incidence of approximately 0·4%, being about the same as that at Broughton Hall Psychiatric Clinic. Similar experience has been noted in the United States of America. There Thompson and McGinnis (1944) found that of the 61,795 patients admitted to the Los Angeles Psychopathic Hospital over a period of seventeen years, 174 (0·28%) were suffering from the results of head injury. These figures indicate the relative rarity of severe chronic psychiatric sequelæ of head injuries. The out-patient psychiatrists at our big public hospitals do not see many of these patients. Perusal of 435 psychiatric case histories of patients seen at Sydney Hospital during 1954, and selected at random, revealed eight cases in which previous head injury was a factor, that is about 2%. In addition, the psychiatrist is occasionally asked to see such patients in consultation in the wards. Repatriation and child guidance clinic psychiatrists see few of these patients. Actually, most patients referred for psychiatric opinion find their way to psychiatrists in private practice, because factors of insurance and of compensation are frequently involved.

The psychiatrist meets the following types of cases (Bowman and Blau, 1949, slightly modified):

> Primary traumatic mental disorders (due in the first place to organic brain damage secondary to head trauma): (i) acute and subacute confusional states; (ii) chronic conditions: (*a*) post-traumatic personality disorders, (*b*) post-traumatic defect conditions, (*c*) mixed types; (*d*) traumatic encephalopathy of pugilists, (*e*) post-traumatic convulsive disorders.
>
> Secondary traumatic mental disorders (associated with, precipitated by, or complicated by, head trauma): (i) psychoneurosis with head trauma; (ii) psychosis with head trauma.

These will now be considered in turn.

Acute and Subacute Confusional States.

In acute and subacute confusional states the essential feature is a state of altered consciousness which is thought to be caused usually by diffuse brain damage immediately following trauma to the head. The symptoms vary from a dazed condition, which may last for a few minutes, to coma with subsequent delirium, which may last for weeks or months. The concussion, assumed to be due to direct damage to neurons, may be accompanied by contusion, due to direct trauma of supporting vascular structures. This is especially so at the *contre coup* area and at the frontal and temporal poles. There is also possibly further damage, such as laceration. Mayer-Gross, Slater and Roth (1954) follow Symonds in classifying cases as mild, moderate or severe. In mild cases the patient may be merely dazed or unconscious for up to an hour. On waking, he may be alert or he may be drowsy and confused. In most such cases mental symptoms clear within a few hours. There is complete amnesia both for the accident and for a brief period before it (retrograde amnesia). At this point it may be remarked that if the patient recalls the impact of the blow, the occurrence of concussion may be disbelieved, although local brain contusion may have occurred (Richardson, 1951).

In cases of moderate severity the stage of coma, which may last several hours, is followed by a stage of delirium. This is thought to be due to cerebral irritation, which may last for a few hours or for a few days. The patient is grossly confused, disorientated, hallucinated, resistive, restless, bewildered and anxious. Occasionally he is violently aggressive or even homicidal. In adults this stage is often followed by a Korsakov or amnesic-confabulatory syndrome, in which the patient is euphoric and facetious if humoured, but sullen and irritable if disturbed or questioned, and still disorientated with recent amnesia and confabulations. This stage is rarely seen in children, probably because their higher perceptive functions are incompletely developed and they are more likely to show emotional changes and impulsiveness. Other types of syndrome sometimes seen at this stage are twilight, fugue or dream states, with complete amnesia for the episodes, and an "apathetic syndrome" in which the patient just lies inert but without clouding of consciousness.

In severe cases coma lasts for several hours or days, and the stages described above may last for days or weeks. In all cases the psychiatrist will be just as alert as the neurosurgeon for the development of neurosurgical complications, such as subdural or extradural hæmorrhage. In general, the shorter the initial disturbance of consciousness, the better the prognosis. In most cases, once consciousness has been regained, the ultimate outlook is good. But convalescence may be marred for some weeks or months by the post-concussion syndrome to be discussed later. Mayer-Gross, Slater and Roth (1954) quote Ritchie Russell's valuable method of assessing the severity of the brain injury and the likely period of incapacity from work, by means of the duration of post-traumatic amnesia. This is measured from the time of injury to the time of the beginning of continuous full awareness, and will usually include the delirious and Korsakov phases. They quote Cairns's average figures (Table I) for service patients (civilian figures show slightly shorter periods of disability):

Table I.

Period of Post-traumatic Amnesia.	Time Absent from Work.
5 minutes to 1 hour.	4 to 6 weeks.
1 to 24 hours.	6 to 8 weeks.
1 to 7 days.	2 to 4 months.
Over 7 days.	4 to 8 months.

The prognosis is worse with increasing age, with alcoholism and in the case of patients with arteriosclerosis.

Treatment of these early stages seldom rests primarily with the psychiatrist. But if problems of management arise he may suggest that certification should be avoided if possible and that paraldehyde or chloral is probably the best sedative. Changes in the electroencephalogram, such as abnormal slow activity, usually, though not always, subside gradually as clear consciousness returns.

[1] Read at a meeting of the Special Group of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association, May 19, 1955.

The Post-Concussion Syndrome.

The post-concussion syndrome is included among the chronic primary post-traumatic personality disorders. But there is considerable difference of opinion as to the relative importance of organic and psychogenic factors in its ætiology. Mayer-Gross, Slater and Roth (1954) believe that underlying this syndrome there is probably some subtle structural modification in the brain which is potentially reversible. But these workers consider that the duration and severity of the symptoms are largely dependent upon the patient's predisposition and current psychological stresses. In favour of an organic basis are the facts that the subjective complaints are remarkably uniform in persons of diverse social and economic strata. Also doctors who have had head injuries themselves become converted from the psychogenic to the organic school of thought. On the other hand, the syndrome may follow quite trivial head injuries with little disturbance of consciousness. The symptoms closely resemble those seen in pure neuroses. The syndrome is common after head injury, and some 55% of patients show signs of it at some stage during their convalescence (Mayer-Gross, Slater and Roth, 1954). It usually becomes manifest as the confusional stage clears up, or when some mental or physical exertion is first attempted in convalescence (Denny-Brown, 1943). Richardson (1951) puts the position very aptly: "The impersonal problem of an unconscious or confused patient with brain injury has changed to a very personal one in which all the individual psychological and social aspects must be considered." The symptoms are headache, dizziness, physical and mental fatigability, poor concentration, irritability, defective emotional control, insomnia, lowered tolerance to alcohol, anxiety, hypochondriasis, mental retardation and lack of initiative. In mild cases these symptoms may not be very obvious while the patient is in hospital, but they are quickly noted by the relatives when he returns home. Rowbotham (1949) paints a vivid picture of the situation in the home when the previously even-tempered, industrious husband returns after his head injury to make life miserable for himself and his wife and children. However, the tendency is towards gradual recovery in periods varying from two weeks to two years. Hence one should refrain from giving a prognosis in some cases for at least two years.

On the other hand, the symptoms of the post-concussion syndrome may worsen or persist, and if this happens it is usually due to psychogenic factors. In other words, a neurotic "overlay" develops. This is especially likely to occur if the patient already has a neurotic predisposition, and if he has psychological and environmental problems such as domestic or financial stress. Possibly his head injury occurred while he was in the midst of some difficult life situation which was already straining his capacity for adjustment to its limits. Moreover, the head is an important part of the body image. Most people know that it contains the brain, which is the seat of their mental life, and many think their "real self" is somewhere behind the eyes. Thus an injury to the head is a special threat to the patient's integrity and feeling of security. Undue anxiety easily develops, only to perpetuate the symptoms already present. A symptom such as dizziness readily feeds a feeling of insecurity. Hope of financial gain from compensation is another factor and usually operates more or less unconsciously. Thus post-traumatic neuroses are far more common in industrial than in amateur sporting accidents, and are more common in the insured than in the uninsured. But true malingering is rare.

How, then, can we estimate the relative contribution of organic and psychogenic factors in the individual case? This is an important problem, and sometimes difficult to solve. Not many cases are mainly organic or mainly psychogenic. In most, both factors play a part in varying proportions. Factors which indicate an organic ætiology are: (i) The injury was moderate or severe. (ii) Neurological signs are present. (iii) There is objective evidence of consistent impairment of memory and intellectual efficiency. (iv) The symptoms show little response to change in surroundings. (v) There is gross intolerance to heat and alcohol. Factors which indicate a psychogenic ætiology are: (i) The patient has a pre-accident history suggestive of neurotic predisposition. (ii) There are contemporary psychological stresses, or factors such as compensation which favour secondary gain, which could account for a continuation of symptoms. (iii) The symptoms are exaggerated or inconsistent. (iv) The patient may be distracted from his disabilities by changes in his surroundings or circumstances. The electroencephalogram may help, for patients with signs of mainly organic ætiology tend to present focal abnormalities, while in those cases of largely psychogenic origin, if anything, only diffuse changes appear on the record. Richardson (1951), Lynn, Levine and Hewson (1945), Grinker and Weinberg (1949), Bowman and Blau (1949) and other authors favour the use of psychometric tests, such as the Rorschach and the Wechsler-Bellevue scale, as an aid to diagnosis. Perhaps the greatest value of these tests at present is their repetition in order to assess the progress. Mayer-Gross, Slater and Roth (1954) quote Lewis's dictum: "When the diagnosis of organic damage is clinically doubtful, psychological tests cannot as yet be relied upon to supply an unequivocal answer." We must remember, too, that the absence of neurological signs does not exclude organic damage. Also electroencephalographic and radiological abnormalities do not necessarily indicate that the symptoms are due to cerebral pathology. Finally, correct diagnosis is not only important for prognostic and medico-legal reasons, but also for the purposes of treatment. An incorrect assessment by the doctor of organic and psychogenic factors will soon be suspected by the patient. The confidence of the patient in the doctor will thereby be shaken and treatment and rehabilitation hindered.

Occasionally, in severe cases, a post-traumatic personality disorder of organic origin persists. The symptoms are labile affect, irritability and impulsiveness. The patient ceases to care about his family and is liable to sudden severe outbreaks of rage, possibly due to damage in the thalamic or prefrontal orbital areas. A few patients show extreme apathy and withdrawal, perhaps due to severe injury of the prefrontal areas.

Children in general stand up to head injuries better than do adults; but occasionally there develops a post-traumatic personality disorder resembling the post-encephalitic type. This disorder is characterized by negativism, restlessness, tantrums, impulsive aggressiveness, delinquency, and poor concentration and performance at school. They may appear to be feeble-minded, but on careful testing their intelligence quotient is usually found to be average. The prognosis is poor, and admission to an institution may ultimately be necessary. Byrne (1951) has reported an interesting case of post-traumatic disturbance of hypothalamic function in a child.

Post-Traumatic Defect Conditions.

Post-traumatic dementia is rare, especially in young subjects. It is associated with severe injuries resulting in loss of neurons, glial reaction, meningo-cortical adhesions and contraction of scars. Radiological examination may reveal cerebral atrophy and ventricular enlargement, and electroencephalography may reveal persistent abnormalities. Post-traumatic dementia is commoner in the elderly, the arteriosclerotic and the alcoholic. The main symptoms are recent and remote amnesia, poor concentration, diminished powers of abstraction and spontaneity of thought (Goldstein, 1952), and emotional lability. Institutional care may be advisable. It should be noted that mild symptoms of this nature which may pass unnoticed in a labourer can cause substantial disability in professional and intellectual workers. Cases of mixed intellectual and personality deficit occur. In children the question of the relationship of head injury to mental defect is often raised by parents. However, Bowman and Blau (1949) estimate that only about 1% of institutional cases of mental defect can be ascribed to brain injury sustained after birth. In other words, a true causal relationship is uncommon. More often, head injuries complicate or exaggerate an already existent mental deficiency. An important

point is that in both adults and children, when standard intelligence tests and deterioration tests, such as the Babcock scale, are being used, allowance must be made for the lack of interest, initiative and concentration, and mental fatigability of the post-concussion syndrome. These symptoms may produce a picture of apparent rather than of real defect, and are likely to clear up in a matter of weeks or months.

In addition to generalized defect conditions, special types of defect may occur, such as aphasia, agnosia, anomia and apraxia. Careful detailed examination is often needed to detect them, but their recognition is important because they complicate the psychological picture. Investigation of these defects may suggest practical methods of rehabilitation by special teaching and occupational therapy, which often bring about slow improvement. They are usually due to lesions in the temporo-parietal area.

The traumatic encephalopathy of pugilists is the "punch-drunk" syndrome, characterized by fatuous euphoria, lack of insight, slurred speech, unsteady gait, and deterioration of attention, memory and boxing skill. It is due to frequently repeated brain injuries causing multiple petechial hæmorrhages in the deeper portions of the cerebrum, followed by degenerative processes and reparative gliosis. It is more common in second-rate fighters used for training purposes and knocked out several times a day. The symptoms progress for a year or so, but become stationary when the patient gives up boxing (Bowman and Blau, 1949).

Post-traumatic epilepsy has been ably discussed by my neurological colleagues. We turn now to the secondary traumatic mental disorders. These exist quite independently of head injuries, but sometimes the question arises as to whether, in a particular case of neurosis or psychosis, head injury is a precipitating, aggravating or contributing agent. The point may be raised either by relatives who feel that the head injury removes the stigma of a family history of mental illness, or by the courts in compensation cases. In some patients the head injury does seem to have some causal relationship to the mental illness, in some there is obviously no such relationship, and in others the issue is doubtful. It seems to be generally agreed among psychiatrists, and upheld by some judicial decisions, that a patient is entitled to compensation when the relationship of a head injury to the development of a mental disorder has been proven. I would draw your attention to Ebaugh and Benjamin's criteria for reaching an opinion in these cases (quoted by Bowman and Blau, 1949):

1. The trauma is a true causative factor when the patient showed no manifest or latent signs of mental illness before the trauma, and to the best of our knowledge mental illness could not have occurred without the trauma.

2. The trauma is a major precipitating factor when it alone could not have caused the mental illness, which is already present in latent form, or for the appearance of which other ætiological factors are necessary. To the best of our knowledge, the mental illness would not have appeared at the time without the trauma, and might never have become manifest at all.

3. Trauma is an aggravating factor when the mental illness is already manifest, but its clinical course is materially affected by the trauma.

4. The trauma is a minor precipitating factor when the mental illness is already present in latent form and, to the best of our knowledge, would have become manifest without the trauma. It is probable that the trauma, either through its physical or psychological effects, is at least partially responsible for the outbreak of the mental illness at the time.

5. The trauma has no demonstrable relation to the mental illness when there is not some continuity of symptoms in chronological relation to the injury, and if there was an asymptomatic period of more than one to three months after the injury (except in cases of post-traumatic epilepsy), or when the trauma is so slight as not to cause unconsciousness or delirium.

Psychoneurosis with Head Injury.

Psychoneurosis with head injury can be dealt with only very briefly in the time available. I have already indicated how the post-concussion syndrome may become the nucleus for neurotic attitudes. This may be expressed in terms of Freud's principle of "somatic compliance", that is, the readiness of psychogenic attitudes to express themselves in symptoms referable to an organ at present or previously diseased. Schilder (1949) feels that traumatic neurosis after head injury should be differentiated from that following other injuries, firstly because head injury produces changes in cerebral mechanisms facilitating neuroses, and secondly because of the particular psychological value the individual places on his head, involving the protection of the ego against an overwhelming stimulus. The neurosis may be interwoven with the organic sequelæ of the head injury, or may develop weeks, months or even years later. We diagnose a traumatic neurosis only when a decided change has taken place in the individual following an injury, and not when the trauma has been a mere insignificant episode in a neurotic's life history. Schilder (1949) distinguishes the following types of reaction: (i) Reactions in which terror or anxiety predominates. (ii) Reactions of neurasthenic and hypochondriacal character. These develop particularly when the body image has been changed in some way, as after a trephine. Repair of the bony defect may be of great psychological value. (iii) Reactions of hysterical type, in which the conversion symptoms, often based on the principle of somatic compliance, include headaches, pareses, anæsthesias, "black-outs", psychogenic amnesia and fugues, and "*la belle indifférence*". (iv) Reactions in which social and compensation problems prevail. The patient demands help and makes it clear to the community that he suffers. Prolonged litigation makes adjustment more difficult, but early and adequate financial settlement, though it helps, does not necessarily end the neurosis.

Psychosis with Head Injury.

As Mayer-Gross, Slater and Roth (1954) assert, it is rare for a true endogenous psychosis of manic-depressive or schizophrenic type to commence after a head injury. When it does, the trauma merely makes overt a predisposition which would probably have become manifest ultimately without traumatic intervention. The psychosis usually develops immediately after the acute organic syndrome. Head injury may also aggravate a preexisting endogenous psychosis, for example when the trauma is the result of a suicidal attempt, in which case symptoms due to the brain trauma may complicate the symptomatology of the psychosis itself. Schizophrenia-like syndromes are occasionally seen in toxic and other organic psychoses, including the traumatic, probably because the organic lesion has mobilized schizophrenic mechanisms in a schizoid personality, and not because the lesion as such can cause specific schizophrenic symptoms.

It is interesting to note that general paresis sometimes becomes manifest shortly after a head injury. Some authors think this is more than a coincidence and that the head injury activates a latent syphilitic process.

Head injury is often associated with alcoholism, and it is often hard to decide whether the case is a true primary traumatic psychosis aggravated by alcoholism or an alcoholic psychosis coincident with or aggravated by head injury. It should be remembered that *delirium tremens* can be precipitated by a head injury just as by other illnesses or operations. In general, chronic alcoholics do not tolerate head injury very well. Cerebral arteriosclerosis, in which rupture of sclerosed vessels with hæmorrhages is more likely than with normal vessels, is often a factor in the development of traumatic dementia.

Prognosis and Treatment.

Some points in prognosis have already been mentioned. In cases of traumatic dementia and organic personality deterioration, rare in the young but commoner in the middle-aged and elderly, the prognosis is grave. In the post-concussional syndrome uncomplicated by permanent

organic damage, factors such as a stable pre-traumatic personality, good native intelligence, real incentives to get well, and prospects of a good domestic and occupational environment after convalescence are far more important for a good prognosis than the severity of the injury (Mayer-Gross, Slater and Roth, 1954).

Rehabilitation should commence as soon as the patient recovers consciousness. At this time he is often highly suggestible and can readily be made anxious about himself and his future by tactless questioning and conversation. He should not be asked about symptoms of which he does not spontaneously complain. Rather should we make use of his suggestibility and surround him with a positive and optimistic atmosphere, giving him reassurance and explanation when he is ready for it. The ideal is that each patient should have a carefully planned regime of occupational therapy, games and exercises involving a gradual increase in his physical and mental activities, care being taken not to over-tax his strength in the early stages. This should be combined with a psychotherapeutic approach to his feelings and problems, and this demands intimate *rapport* with him. Depth psychotherapy is rarely required, though Klingman (1945) and Grinker and Weinberg (1949) found narcoanalysis useful occasionally. Sympathetic explanation, guidance and advice usually suffice. Lectures, group discussions and group psychotherapy have been found useful. An interesting development is the establishment of rehabilitation centres, such as Ritchie Russell's (1949) head injury advice bureau at Oxford staffed by a neurologist and a social worker especially experienced in head injuries. It is claimed that such centres can prevent much chronic invalidism. The patient's return to work may have to be gradual, and for some a change of occupation and a course of retraining will be advisable. The earlier a patient can be returned to work, the better, and compensation negotiations should be concluded as soon as possible. Rowbotham (1949) and others favour early lump sum settlement. Speech therapy may be necessary for persistent dysphasia.

Some patients with head injury are accident-prone, and inquiry will reveal that they have had repeated accidents in the past. They often have a specific character structure — for example, over-anxious types or over-confident, careless types, or restless, hyperkinetic children. Some seem to seek accidents unconsciously as self-punishment. In other words, psychological factors may have played a part in the genesis of the accident, and one should always inquire about this. If a patient has repeated accidents in a particular occupation, it may be advisable to change to a less risky one. Finally, the psychiatrist occasionally meets with the problem of the advisability or otherwise of electroplexy or deep coma insulin therapy in endogenous psychoses precipitated by head trauma. I have been unable to find an authoritative statement on this; but I think that more conservative measures should be tried first, and if they fail one should ensure by means of a competent neurological assessment that any organic lesions are thoroughly healed before one adds the possibility of further brain trauma by energetic physical therapies.

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TEN CASES OF RHEUMATOID ARTHRITIS TREATED WITH PAS AND OTHER MEASURES OVER A TWO-YEAR PERIOD.

By Brian Haynes,
Sydney.

PAS (para-amino salicylic acid) was first given to patients suffering from rheumatoid arthritis at the Saint Vincent's Hospital Rheumatism Clinic in November, 1952, after favourable reports had been published by Brous (1952). Previously, Duthrie and Swanson (1948) had reported that PAS was useless in rheumatoid arthritis. However, reference to their article reveals that it was given in very high dosage, 24 grammes per day, and had to be suspended in a few weeks in most cases on account of toxic symptoms. The number of cases was small (six), and the longest period for which the drug was given was thirty-six days. In one case it was given only for six days.

For some time treatment at the clinic had been standardized to the following plan: (i) rest: general bed rest during systemic activity, local rest with splinting when indicated; (ii) prophylactic administration of sulphadiazine, 0·5 gramme twice a day indefinitely; (iii) the exhibition of some salicylate—sodium salicylate, aspirin, succyl-salicylate and later "Disprin"; (iv) the administration of gold, as "Myocrisin", usually in injections of 0·05 gramme weekly, fortnightly or monthly. In accordance with Brous's report, it was decided to use PAS as the salicylate for a period. The early patients appeared to respond better than had been our experience in the previous ten years. The following ten patients were set aside to follow over as long a period as was feasible. In five cases no gold was given, the drugs being restricted to sulphadiazine, given prophylactically, and PAS, 0·5 gramme every four hours or 1·0 gramme every four hours.

The patients were presented at the clinical evening of the New South Wales Branch of the British Medical Association at Saint Vincent's Hospital in 1954. My findings and conclusions were later published in this journal in the form of a letter. In the subsequent correspondence it was requested that the results should be classified according to the classification of the American Rheumatism Association, and reviewed after a period of two years. So they are presented again, and an attempt has been made to classify the therapeutic response according to the American formula (see appendix).

Of the 10 patients, the one who has been longest under treatment with PAS has been taking the drug continuously for thirty-one months; the shortest period has been fourteen months. The usual dosage has been three grammes per day. This has been the minimum dosage. The maximum dosage has been six grammes per day. In

TABLE I.
Evaluation of Ten Patients for First and Second Year.

Case Number.	Patient.	Age. (Years.)	Occupation.	Onset.	PAS Therapy.	Classification.	Therapeutic Response.	
							July, 1954.	July, 1955.
I	Mrs. A.	45	Housewife.	1943.	February, 1952.	Stage 2, Class 3; B.S.R., 25 millimetres; steady deterioration.	Class 2, major improvement; B.S.R., 7 millimetres; all home duties.	Class 1, complete remission; B.S.R., 8 millimetres; all home duties.
II	Mrs. B.	33	Housewife (2 children).	1950.	April, 1953.	Stage 2, Class 3; B.S.R., 28 millimetres.	Class 1, complete remission; B.S.R., 10 millimetres; all home duties.	Class 2, major improvement; B.S.R., 19 millimetres; all home duties.
III	Miss C.	40	Staff sister.	June, 1953.	July, 1953.	Stage 1, Class 4; B.S.R., 56 millimetres.	Class 1, complete remission; B.S.R., 2 millimetres.	Class 1, complete remission; B.S.R., 5 millimetres.
IV	Mr. D.	74	Hairdresser.	1950.	August, 1953.	Stage 2, Class 3; B.S.R., 34 millimetres.	Class 1, complete remission; B.S.R., 10 millimetres.	Class 1, complete remission; B.S.R., 11 millimetres.
V	Mr. E.	40	Warder.	February, 1953.	October, 1953.	Stage 1, Class 3; B.S.R., 18 millimetres.	Class 1, complete remission; B.S.R., 2 millimetres.	Class 1, complete remission; 4 millimetres.
VI	Mrs. F.	40	Waitress.	February, 1953.	December, 1953.	Stage 1, Class 3; B.S.R., 52 millimetres.	Class 2, major improvement; B.S.R., 13 millimetres.	Class 1, probable complete remission; B.S.R. unprocurable.
VII	Miss G.	56	Cleaner.	September, 1953.	January, 1954.	Stage 1, Class 3; B.S.R., 38 millimetres; bronchiectasis.	Class 1, major improvement; B.S.R., 27 millimetres.	Class 1, major improvement; B.S.R., 29 millimetres.
VIII	Mrs. H.	50	Pensioner.	1943.	January, 1954.	Stage 4, Class 4; B.S.R., 40 millimetres; constant pain.	Class 3, major improvement; B.S.R., 29 millimetres; free from pain.	Class 3, major improvement; free from pain.
IX	Mr. I.	66	Labourer.	July, 1953.	February, 1954.	Stage 2, Class 3; B.S.R., 26 millimetres.	Class 2, major improvement; B.S.R., 20 millimetres; old age pension.	Class 1, complete remission; B.S.R., 9 millimetres; old age pension.
X	Mr. J.	61	Wharf labourer.	1950.	May, 1954.	Stage 4, Class 4; B.S.R., 60 millimetres; 9 stone 7 pounds; emaciated; constant pain.	Class 3, major improvement; B.S.R., 48 millimetres; 10 stone 9 pounds; free of pain.	Class 3, major improvement; B.S.R., 33 millimetres; 11 stone 13 pounds, normal weight; free of pain.

no case has the drug had to be suspended. There have not been any toxic symptoms. All the patients are still alive. In this regard the series is fairly representative of all grades of the condition, three patients having been acutely and dangerously ill at the beginning of treatment (Cases III, VI and X).

Eight of the patients are back at their usual occupations, and of these eight, seven are free of pain or disability. The patient (Case II) who has periodic attacks of pain in her feet or one finger for three or four days at a time, suffered a miscarriage, from the effects of which she has not as yet made a complete recovery. She is the only patient in the series to whom additional drug therapy ("Butazolidin") has been given.

Five patients have normal blood sedimentation rates. A sixth patient (Case VI) has been asymptomatic for over a year and is working as a shop assistant in Victoria. A year ago her blood sedimentation rate was 13 millimetres in one hour. It may well be normal. A seventh patient (Case VII) has been asymptomatic for over a year, working as a cleaner. When her arthritis was at its height her blood sedimentation rate was 38 millimetres in one hour. Since she has been asymptomatic her blood sedimentation rate has been steady at from 25 to 30 millimetres in one hour. She also suffers from bronchiectasis, which may well be responsible for the residual rise in her blood sedimentation rate. In every case the blood sedimentation rate is appreciably lower than when treatment was begun.

In eight cases there is a complete, pain-free range of movement in every joint. In no case has subluxation, ulnar deviation or hyperextension developed. The only patients (Cases VIII and X) with deformities and limited joint function had greater loss of function and irreparable damage before their treatment was commenced.

In five cases (I, IV, VII, IX and X) no gold has been given during the past year. In three cases (IV, VIII and IX) gold was not given at all, the only drugs being sulphadiazine given prophylactically and PAS.

An assessment of the therapeutic response according to the criteria of the American Rheumatism Association is given in Table II. Five patients, possibly six, have achieved a complete remission — Grade I therapeutic response. The remainder have made a major improvement—Grade II therapeutic response.

After a further twelve months' observation of these 10 patients and of some 60 other patients under similar treatment I am convinced that PAS has an antirheumatic action similar to the antirheumatic action of other salicylates such as sodium salicylate and aspirin. Its action is slower than that of sodium salicylate or aspirin, but more prolonged. The slowness of its action appears to be a possible reason for its disfavour with many. In rheumatoid arthritis both patients and doctors are impatient. They hope for and expect recovery in days or weeks when they are dealing with a disease that moves in terms of months. It is essentially a disease of bone and cartilage as well as systemic disease. Even a simple fracture takes a certain time for repair, and it is unlikely that any drug will ever appreciably hasten the process. I think the slower *tempo* of PAS is an advantage in rheumatoid arthritis. The deleterious effects of the fast-acting drugs, such as cortisone, are all too familiar. Relieved of his pain, the patient is up and about, subjecting his unhealed joint surfaces to further trauma.

In the correspondence following my first report, Highton (1955) wrote as follows:

> I would like to make a further point, that in the assessment of any therapeutic agent in this disorder it it is essential to test it over a prolonged period of time (at least two years in my opinion), in order to try to control the variable factor of spontaneous remissions and exacerbations.

This did not prevent him from expressing an adverse opinion in the same paragraph from "a very small pilot series of patients". However, I quite agree with his criteria for assessment. My opinion in the matter may be wrong; but at least I have fulfilled the criteria for assessment and have studied adequate data.

Since the theory was advanced in July, 1954, that PAS and the salicylates act in rheumatoid arthritis through their antihistamine activity (Trethewie, 1951, 1952) and

that the fundamental pathological process is an allergic arteritis of the articular vasculature, Ball (1954) reports finding at post-mortem examination typical lesions of *polyarteritis nodosa* in the visceral vessels of subjects of rheumatoid arthritis. In this thesis *polyarteritis nodosa* is considered to be the malignant type of rheumatoid arthritis. There is a suspicion that severe rheumatoid arthritis can pass into the polyarteritic phase through the administration of cortisone.

Although I think PAS has antirheumatic action, it is not in itself a "cure" for rheumatoid arthritis. In this respect no drug is in itself a cure for rheumatoid arthritis. I doubt whether any such panacea will ever be found. However, I think that these cases illustrate that a comprehensive routine of treatment comprising rest, the use of safe antirheumatic drugs, the prophylactic administration of sulphonamide drugs, patience, psychotherapy and prolonged supervision can prevent the development of crippling deformities and restore the non-neglected patient to normal or nearly normal life in a reasonable period of time.

Reports of Cases.

Case I.—Mrs. A, aged forty-five years, developed pain and stiffness in the left shoulder in 1943, followed by involvement of the proximal interphalangeal joints and wrists. X-ray examination revealed loss of joint space and osteoporosis. Her blood sedimentation rate was 21 millimetres in one hour. She was treated with rest, gold and sodium salicylate. Her illness ran an intermittent course till 1952, the blood sedimentation rate remaining around 20 millimetres in one hour. Her feet became involved also, as well as the metacarpophalangeal joints. In November, 1952, her treatment was changed to rest, gold, PAS and sulphonamides. The blood sedimentation rate was 25 millimetres in one hour and her weight was six stone nine pounds. By February, 1953, she was feeling very well. In May, 1953, her blood sedimentation rate was 12 millimetres in one hour and her weight was six stone thirteen pounds. In September she developed a mild rash and the gold therapy was suspended. She has not had any gold since. With sulphonamides and PAS only for the past twenty-two months she has remained well, with a normal blood sedimentation rate and minimal enlargement of one interphalangeal joint, but full movement, and she is able to carry out her household duties.

Case II.—Mrs. B., aged thirty-three years, developed polyarthritis in 1950, with typical involvement of the metacarpophalangeal joints, wrists, elbows, shoulder girdle and knees. Her blood sedimentation rate was 35 millimetres in one hour and the hæmoglobin value was 10·4 grammes per 100 millilitres. The course of her illness had been up and down, with gradual deterioration in 1951 and 1952. Treatment was started in April, 1953, with rest, "Myocrisin", prophylactic administration of sulphonamide and PAS. Steady progress was maintained till July, 1954, when she was coping with her wifely and maternal duties (two children). Her blood sedimentation rate was 10 millimetres in one hour. A minor remission occurred in November, 1954, and "Butazolidin" (100 milligrammes twice a day) was added. Her blood sedimentation rate was 26 millimetres in one hour after her miscarriage. The blood sedimentation rate was 19 millimetres in one hour in July, 1955. She has nearly full, painless movement of all joints and no deformity. She was emotionally upset for one month because her son was in hospital for a strabismus operation. Her feet and hands are painful for several days at a time with barometric changes.

Case III.—Miss C, a senior sister in the out-patient department, developed acute polyarthritis with typical involvement of all joints, the lower part of the spine and the hips excepted, in June, 1953. She was toxæmia and febrile, with a blood sedimentation rate ranging from 30 to 50 millimetres in one hour for upwards of six months. The standard treatment was started in July, 1953. She was asymptomatic and deformity-free by February, 1954, and resumed part-time work shortly after. By July, 1954, her blood sedimentation rate was two millimetres in one hour; but I graded her as "Class 2 functional capacity, Major improvement, grade 2 Therapeutic Response". The past year has passed with minor incidents following over-use of single joints. She is still doing full-time duty and is asymptomatic with no deformities. Her blood sedimentation rate is five millimetres in one hour. She could probably be classed as having a "complete remission, grade I therapeutic response".

Case IV.—Mr. D, aged seventy-four years, a hairdresser, developed polyarthritis of the hands and feet, with typical rheumatoid distribution, in January, 1950. His blood sedimentation rate was 34 millimetres in one hour. From August, 1953, when he was started on the standard treatment with rest, sulphonamides and PAS, he has made a satisfactory improvement. By July, 1954, he was largely asymptomatic, with full painless movement and no deformity, and has been back at work most of the year. The past year has been the same. In July, 1955, his blood sedimentation rate was 11 millimetres in one hour. He could be graded as "Class I Functional Capacity with a complete remission, Grade I Therapeutic Response".

Case V.—Mr. E, aged forty-one years, a gaol warder, developed polyarthritis of the heels, knees, elbows, hands and shoulders in October, 1953. His blood sedimentation rate was 25 millimetres in one hour. Treatment with rest, gold, sulphonamides and PAS was started in October, 1953. He made a rapid improvement and was back at work by March, 1954. He has not lost any time through illness since. He has a full range of painless movements in all joints. There is no deformity. His blood sedimentation rate in July, 1955, was four millimetres in one hour. He has full functional capacity and can be classed as having a complete remission (first-class therapeutic response).

Table II.

Therapeutic Response.

Grade.	July, 1954.	July, 1955.
I. Complete remission.	4 cases.	5 cases.
II. Major improvement.	6 cases.	5 cases.
III. Minor improvement.	Nil.	Nil.
IV. No improvement or progression.	Nil.	Nil.

Case VI.—Mrs. F, aged forty-one years, developed pain over the left sacro-iliac joint in November, 1953. A week later both knees became swollen and painful, and this was followed by affection of the metacarpo-phalangeal joints of both hands and the wrists. She became nauseated and began vomiting after each meal. She had typical metacarpophalangeal involvement. The blood findings were as follows: the erythrocytes numbered 4,200,000 per cubic millimetre, the hæmoglobin value was 12·3 grammes *per centum*, and the blood sedimentation rate was 52 millimetres in one hour. Treatment with rest, gold, sulphonamides and PAS was started in January, 1954. By July, 1954, she had been back to work as a waitress for two months (against advice), but was feeling well. Her blood sedimentation rate was 18 millimetres in one hour. Later she moved to Victoria. In July, 1955, it was ascertained that she was working as a shop assistant and was symptom-free.

Case VII.—Miss G, aged fifty-six years, developed polyarthritis affecting the metacarpo-phalangeal joints, wrists, shoulders and one knee, in September, 1953. She had suffered from bronchiectasis for years. Her blood sedimentation rate was 32 millimetres in one hour. Treatment was started in January, 1954. She was asymptomatic and back at work after four months. In July, 1954, her blood sedimentation rate was still raised (20 millimetres in one hour) and it has remained about this level, despite her otherwise fairly complete remission. The bronchiectasis may well play a major part in the raised blood sedimentation rate. Her condition is assessed as "Functional Capacity, Class I, with Grade 2 (major improvement) Therapeutic Response".

Case VIII.—Mrs. H, aged fifty years, developed rheumatoid arthritis in 1943. She was bedridden with advanced, nodular type rheumatoid arthritis and gross deformities when her treatment was commenced in January, 1954. Her blood sedimentation rate was 40 millimetres in one hour, and she was in constant pain. Her treatment consisted of rest, sulphadiazine (0·5 gramme twice a day) and PAS (0·5 gramme every four hours). After three months she was practically free of joint pain. In July, 1954, her blood sedimentation rate was 29 millimetres in one hour. She was still free from pain for the greater part of the time in July, 1955. Her blood sedimentation rate was 37 millimetres in one hour.

Case IX.—Mr. I, aged sixty-six years, a labourer, developed acute polyarthritis of both hands in August, 1953. When first examined in February, 1954, he had typical involvement of the metacarpo-phalangeal and proximal interphalangeal joints of both hands. His blood sedimentation rate was 26 millimetres in one hour. X-ray examination revealed decalci-

fication (juxtaarticular) and narrowing of the joint spaces. Treatment with rest, the prophylactic administration of sulphadiazine and PAS was begun. By July, 1954, he was free of pain, with no deformities and full, painless movement of all joints. His blood sedimentation rate was 20 millimetres in one hour. In November his blood sedimentation rate was nine millimetres in one hour and he was referred to the occupational therapy department for light work. He has not attended since; but in July, 1955, the condition of his hands was perfect as regards pain, structure and function.

CASE X.—Mr. J, aged sixty-one years, developed pain in both feet in 1950. A year later he developed pain and stiffness in the right hand. Later the elbows and knees were involved. He consulted a doctor, but was told that not much could be done for him. After a fall on the buttocks he developed two painful tumours over the ischial tuberosities; these were removed surgically, but the sinuses had not healed. He had gross deformity and stiffness of most of the small joints of the hands, feet, knees and elbows. The erythrocytes numbered 3,200,000 per cubic millimetre, the hæmoglobin value was 8·7 grammes *per centum*, and the blood sedimentation rate was 60 millimetres in one hour. He was emaciated and weighed ten stone. Treatment with rest, gold, sulphonamides and PAS was started in May, 1954. By November he was practically free of pain and had gained one stone thirteen pounds. His blood sedimentation rate was down to 48 millimetres in one hour. By December he weighed twelve stone six pounds, and in June, 1955, he weighed fourteen stone, his normal weight. The blood sedimentation rate was 33 millimetres in one hour and the hæmoglobin value was 14 grammes *per centum*. There has been considerable improvement in the movements of his joints, but bony deformity had occurred before treatment was started.

Summary.

1. Ten patients suffering from rheumatoid arthritis have been followed for fourteen to thirty-one months on a standard treatment.

2. The standard treatment comprised rest, the prophylactic administration of sulphadiazine, and the exhibition of PAS and gold.

3. The results have been assessed according to the criteria of the American Rheumatism Association.

4. I consider that PAS has antirheumatic activity.

Acknowledgements.

I am indebted to Dr. A. Sheehan and Dr. P. J. McMahon for their assistance in supervising these patients; also to the pathology department, Saint Vincent's Hospital, for the numerous pathological tests carried out.

Addendum.

A further explanation for inconsistent findings with PAS in rheumatoid arthritis is the fact that a number of preparations available are completely unsatisfactory. Repeatedly patients have brought me chocolate-coated tablets which have passed through in their motions. Obviously there was no absorption. Checking with the Anti-Tuberculosis Association authorities, I found that they had had similar difficulties. It is advisable to prescribe the uncoated tablet of a reliable firm.

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Appendix.[1]

TABLE I.

Classification of Rheumatoid Progression.

Stage	Roentgenologic Signs	Muscle Atrophy	Extra-Articular Lesions (Nodules Tenovaginitis)	Joint Deformity	Ankylosis
I	Osteoporosis sometimes no destructive changes	0	0	0	0
II	Osteoporosis slight cartilage or subchondral bone destruction may be present	Adjacent	May be present	0	0
III	Osteoporosis cartilage destruction bone destruction	Extensive	May be present	Subluxation ulnar deviation and/or hyperextension	0
IV	Same as III with bony ankylosis	Extensive	May be present	Same as III	Fibrous or bony ankylosis

[1] From Steinbroker, Traeger and Batterman (1949).

TABLE II.

Classification of Functional Capacity.

Class

I Complete
Ability to carry on all usual duties without handicaps

II Adequate for normal activities
Despite handicap of discomfort or limited motion at one or more joints.

III Limited
Only to little or none of duties of usual occupation or self care

IV Incapacitated, largely or wholly
Bedridden or confined to wheelchair; little or no self care

Therapeutic Criteria for Response of Rheumatoid Activity.[1]

To provide the most convenient and reliable means of assaying results, four categories of response of rheumatoid activity to treatment are recommended. These range from grades I to IV. Grade I represents maximum improvement; grade IV no improvement, and grades II and III intermediary stages. The criteria are based entirely on objective evidence, since subjective symptoms are considered unreliable.

Grade I, Complete Remission

*1. No systemic signs of rheumatoid activity.
*2. No signs of joint inflammation.
*3. No evidence of activity in any extra-articular process, including nodules, tenovaginitis and iritis.
*4. No remaining impairment of joint mobility, other than that associated with irreversible changes.
*5. No elevation of erythrocyte sedimentation rate.
6. Articular deformity, or extra-articular involvement due to irreversible changes, may be present.

Grade II, Major Improvement

*1. No systemic signs of rheumatoid activity with exception of an elevated sedimentation rate and vasomotor imbalance.
*2. Major signs of inflammation resolved, such as heat, redness of joints and of extra-articular involvement.
*3. No new rheumatoid process of intra-articular or extra-articular structures.
4. Minimum joint swelling may be present.
5. Impairment of joint mobility associated with minimum residual activity may be present.
6. Articular deformity, or extra-articular involvement due to irreversible changes, may be present.

Grade III, Minor Improvement

Any decrease in signs of rheumatoid activity inadequate to fulfil the criteria of grade II:

*1. Diminution of systemic signs of rheumatoid activity.
*2. Signs of joint inflammation only partially resolved.
*3. No evidence of extension of rheumatoid activity into additional articular or extra-articular structures.
4. Decreased but not minimum joint swelling present.
5. Impairment of joint mobility due to residual inflammation may be present.
6. Articular deformity, or extra-articular involvement due to irreversible changes, may be present.

Grade IV, Unimprovement or Progression

*1. Undiminished signs of rheumatoid activity, regardless of functional capacity.
*2. Exacerbation of any previously involved joints or joint, or development of new sites of rheumatoid activity.
*3. Roentgenologic changes indicative of progression of the rheumatoid process, excepting hypertrophic changes.
4. In the presence of one or more of the aforementioned criteria, improvement in other features, including a normal or lowered erythrocyte sedimentation rate, not significant.

Application of the Therapeutic Criteria and Supplementary Aids as a System of Classification and Evaluation:

The first consideration in undertaking the treatment of a patient with rheumatoid arthritis is to determine: (1) the stage of the disease, (2) the presence of rheumatoid activity and (3) the degree (class) of functional impairment. A patient would therefore be classified as having rheumatoid arthritis, active or inactive, stage —, class —.

[1] The criteria prefaced by an asterisk are those which must be present to permit classification of a patient in any particular stage or grade.

THE HEAF MULTIPLE PUNCTURE TEST COMPARED WITH THE MANTOUX TEST IN EPIDEMIOLOGICAL SURVEYS.

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As a result of being actively engaged in the Mantoux testing of fairly large groups of children and adolescents, I felt that it would be opportune to compare the effect and accuracy of the Mantoux test and the Heaf multiple puncture test. Briefly, the standards utilized were as follows. The Mantoux test consisted in the intradermal injection of 10 tuberculin units (0·1 milligramme) of old tuberculin, as this is the standard laid down in Australia for testing prior to B.C.G. inoculation, and the Heaf test was performed in the manner originally described by Heaf (1951). In this method a film of standardized undiluted adrenalized old tuberculin is placed on the skin. The Heaf apparatus is so devised as to introduce the old tuberculin by puncture with a circle of six needles to a regulated depth of one or two millimetres, depending on the texture of the skin. The diameter of the circle is six millimetres, which is the same as the standard diameter of a minimal positive Mantoux reaction. For the purposes of the survey each patient was given both tests, the Mantoux test being performed on the volar surface of the left forearm, and the Heaf test on the volar surface of the right forearm at the same time. The reactions were read seventy-two hours later. All tests were performed and the results read by two operators (doctor and sister) at the inception of the trial and on all group surveys thereafter. However, when the standards had been agreed upon, routine skin-testing and reading of results was carried out by the trained sisters at the Brisbane Chest Clinic.

The trial included subjects of all ages; these consisted of National Service trainees, young children, and adults undergoing routine investigation at the clinic. The cross-section would appear to have a strong preponderance of the first two groups; 95% of those tested were aged nineteen years and under.

TABLE I.
Natural Positive Reactions.

Observation.	Heaf Test (2944 Subjects)	Mantoux Test (2944 Subjects)
Number of negative results	2155	2199
Number of positive results	789 (26·8%)	745 (25·3%)
Ratio of positive results	106	100
Heaf Grade 1, Mantoux 5 to 9 millimetres	407	391
Heaf Grade 2, Mantoux 10 to 14 millimetres	192	205
Heaf Grade 3, Mantoux 15 to 19 millimetres	108	87
Heaf Grade 4, Mantoux 20+ millimetres	82	62

Before I proceed to the statistical analysis, a few comments follow on the apparatus and technique employed. The Heaf multiple puncture apparatus is an instrument which has, in my opinion, many advantages over the tuberculin syringe, and the test thus has advantages over the Mantoux test. These are simplicity of technique and apparatus. Its use is painless and does not frighten children. In addition, human error and skill are not important

factors, and great economy in tuberculin syringes and needles is possible. In mass B.C.G. campaigns the use of relatively unskilled operators in field work is unavoidable in most countries, and the Heaf apparatus provides a solution to operator deficiencies, problems of transport, sterilization of material, and potency of the old tuberculin used in the test. It was found possible with experience to carry out four or five Heaf tests per minute, one cubic centimetre of old tuberculin being used for 250 to 300 subjects. Without the necessity for the careful technique of intradermal injection, little time need be wasted in calming excited children, and rapidity of performance is easily effected. Standards for comparison as originally described by Heaf were accepted; these are tabulated as follows:

Negative:

Minute puncture scars with no erythema.

Minute puncture scars with slight erythema, but no induration.

Positive:

1. Minute puncture scars with definite induration.
2. Coalescence of indurated papules to form a ring of induration.
3. Weal of induration 5 to 10 millimetres in diameter.

Any reaction greater than a weal 10 millimetres in diameter was recorded as "4", and severe reactions as "4+".

These grades were taken to correspond to Mantoux reactions of the following degree (as originally set out for the same purpose by Heaf in 1951):

Heaf Grade 1 equivalent to Mantoux reaction of diameter 5 to 9 millimetres.

Heaf Grade 2 equivalent to Mantoux reaction of diameter 10 to 14 millimetres.

Heaf Grade 3 equivalent to Mantoux reaction of diameter 15 to 19 millimetres.

Heaf Grade 4 equivalent to Mantoux reaction of diameter 20 millimetres or more.

A Mantoux reaction of one to four millimetres in diameter was classified as "negative".

TABLE II.

Positive Reactions after B.C.G. Vaccination.

Observation.	Heaf Test (1052 Subjects).	Mantoux Test (1052 Subjects).
Number of negative results	100	101
Number of positive results	952 (90·5%)	951 (90·4%)
Ratio of positive results	100	100
Heaf Grade 1, Mantoux 5 to 9 millimetres	494	299
Heaf Grade 2, Mantoux 10 to 14 millimetres	337	386
Heaf Grade 3, Mantoux 15 to 19 millimetres	93	212
Heaf Grade 4, Mantoux 20+ millimetres	28	54

Severe reactions have been observed to be relatively uncommon with the Heaf test, a finding contrary to Heaf's original statement in this regard. This feature is an additional advantage of the multiple puncture test, for obvious reasons.

All B.C.G. vaccinations were carried out after a negative response to the Mantoux test had been obtained, and the state of sensitivity to tuberculin was retested two months later by means of the comparative technique described in this article. Again, there are more Grade 1 reactions (494 as opposed to 299) with the Heaf test than with the Mantoux test, though no preponderance in favour of the Heaf test was discovered in the determination of actual positive reactors.

Heaf (1951) found a positive ratio of 113:100 in favour of the multiple puncture test, and Stott (1955) found a ratio of 102:100 in tests on East African adult Negroes. The proportion found in the present survey is 103:100, which represents the total obtained in the following groups: Of all children tested the proportion was 106:100 before B.C.G. vaccination (natural conversion rate—Table I) and 100:100 after B.C.G. inoculation (induced tuberculin sensitivity—Table II). There is therefore more chance of detecting a weakly sensitive individual in the Heaf Grade 1 group than in the minimal positive Mantoux reaction group. It was the experience of the observers that the closely grouped indurated papules as produced by the Heaf instrument were definitely palpable (compare the ease with which Braille type is palpated), whereas the Mantoux reaction of five or six millimetres very often remains a debatable entity.

TABLE III.

Heaf Reaction Grades Compared with Mantoux Reactions in Millimetres.

Test and Reaction.	Natural Reactions.	Reactions After B.C.G.	Totals.
Mantoux 0 to 4 millimetres:			
Heaf Grade 0	2114	59	2173
Heaf Grade 1	77	38	115
Heaf Grade 2	8	4	12
Heaf Grade 3	—	—	—
Heaf Grade 4	—	—	—
Mantoux 5 to 9 millimetres:			
Heaf Grade 0	33	31	64
Heaf Grade 1	264	224	488
Heaf Grade 2	79	42	121
Heaf Grade 3	10	2	12
Heaf Grade 4	5	—	5
Mantoux 10 to 14 millimetres:			
Heaf Grade 0	7	8	15
Heaf Grade 1	53	170	223
Heaf Grade 2	82	161	243
Heaf Grade 3	42	40	82
Heaf Grade 4	21	7	28
Mantoux 15 to 19 millimetres:			
Heaf Grade 0	1	2	3
Heaf Grade 1	12	52	64
Heaf Grade 2	15	110	125
Heaf Grade 3	36	36	72
Heaf Grade 4	23	12	35
Mantoux 20+ millimetres:			
Heaf Grade 0	—	—	—
Heaf Grade 1	1	10	11
Heaf Grade 2	8	20	28
Heaf Grade 3	20	15	35
Heaf Grade 4	33	9	42
Total	2944	1052	3996

It will be noted that the large majority of reactions fall within the expected range of Mantoux equivalents as specified by Heaf (see Figure I).

Discussion.

The problem to be determined at this juncture is the actual significance of the Grade 1 Heaf reaction. The present series seems to indicate that the Grade 1 Heaf reaction shows a true tuberculin sensitivity—that is, a sensitivity to a weak dose of tuberculin, as its close correlation with the occurrence of large numbers of minimal Mantoux reactions is obvious from the tables. It is easy with practice to differentiate the Heaf Grade 1 reaction from puncture marks, trauma and erythema. Tautening the skin will not eradicate true induration, and is advised in all doubtful cases. The impression has been gained that the positive Heaf reaction persists for longer than the Mantoux reaction and may be read with confidence up to ninety-six hours after the test; this allows mothers more latitude in regard to the return visit for reading.

The ease and painlessness of the technique with the use of the Heaf apparatus suggest the possibility of using the Heaf apparatus for actual B.C.G. inoculations, in a

manner similar to the multiple puncture apparatus used by Rosenthal and Birkhaug, though nothing has as yet been done in this regard. B.C.G. of a much greater strength than that at present supplied would probably be required. The working out of a system of both Mantoux testing and B.C.G. inoculation by a simple device requiring little skill, the same instrument being used for both purposes and complicated paraphernalia excluded, would appear to commend itself particularly to large-scale surveys both in civilized communities and in the difficult conditions of field work in under-privileged countries.

FIGURE I.

Summary.

The Heaf multiple puncture tuberculin test was compared with the standard Mantoux test on 1052 persons vaccinated with B.C.G. and on 2944 unvaccinated persons. In both series the Heaf test gave slightly more positive reactions, but fewer severe reactions.

It is concluded that the Heaf test is just as reliable as the standard Mantoux technique and that it has important practical advantages.

Acknowledgements.

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MOLLUSCUM SEBACEUM, SELF-HEALING EPITHELIOMA AND TUMOUR-LIKE KERATOSIS, WITH REPORT OF A CASE.

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IN a review of the available literature of *molluscum sebaceum* (also variously described as kerato-acanthoma and *molluscum pseudocarcinomatosum*), it is notable that there is no record of the fingers being involved by the lesion, and in all cases described the lesion was solitary. Furthermore, in the criteria listed by MacCormac and Scarff (1936), who first described the condition, the face was noted as the site of election and the lesion was described as a single one.

In the condition known as "spontaneously healing epithelioma of the skin", which, macroscopically and histologically, is similar to *molluscum sebaceum* and considered identical by some authors, the lesion is distinguished by being multiple in nature and with a familial incidence in about half the cases described.

There is a third condition, of which only two cases are described in the available literature, called "tumour-like keratosis" (Poth, 1939); this again is macroscopically and histologically similar to *molluscum sebaceum*, and also is considered identical by some authors. However, it differs in that it involves the hands especially, is multiple in character, and follows exposure to sunshine, and possibly also to tar. A case will be described of a similar character to these two cases, and an attempt made to establish the clinical relationship of the three conditions mentioned.

Molluscum Sebaceum.

MacCormac and Scarff (1936), who distinguished *molluscum sebaceum* from squamous carcinoma of the skin, listed ten cases and gave the following six distinctive clinical features, apart from the distinctive histological appearance: (i) The lesion develops rapidly to its maximum in four to six weeks and then remains stationary. (ii) In no case has more than one lesion been present. (iii) The middle area of the face is the site of election. (iv) The lesion is usually of firm consistency, but central degeneration may occur. (v) In all cases the patients are of mature age—generally over sixty years. (vi) Both sexes are affected.

Clinically the lesion is dome-shaped, with a keratin plug projecting through a central hole. Histological examination of sections of a tumour shows a keratin-filled cavity lined by hyperplastic epithelium in papillary projections, with cords and cell nests penetrating the dermis. At the advancing margin, variation in size of the nucleus is seen, and also numerous mitoses occur while the lesion is enlarging.

The largest series was published by Beare (1953), who added a description of 76 cases, which represented about 10% of all the malignant skin conditions seen at the Royal Victoria Hospital, Belfast. These cases conformed to MacCormac and Scarff's clinical criteria in the following ways: (i) Growth was rapid, the duration of the lesion being less than two months in almost 80% of cases. Natural growth seemed to cease at a diameter of about 1·0 to 1·5 centimetres, but spontaneous resolution was seen in only four out of the 76 cases. (ii) In no case was more than one lesion present. (iii) Of the 76 cases, in 64 the lesions were on the face, in 10 on other parts of the head and neck, and in two on the forearm. (iv) The consistency of the lesion was as described by MacCormac and Scarff. (v) Of the 76 patients, only seven were aged under thirty-five years. (vi) The series included 35 males and 41 females. In only four of the cases were there associated actinic keratoses.

The condition has attracted general attention only in the past few years, and various authors have reported small series of illustrative cases.

Spontaneously Healing Squamous-Cell Carcinoma.

Fouracres and Whittick (1953) extracted from the literature 11 cases of spontaneously healing squamous-cell carcinoma. The lesion was first described by Smith (1934). Typically the lesions are multiple, involve the face, trunk and limbs, grow fairly rapidly over a period of three to eight months and then spontaneously regress. The patients involved range in age from twenty-three to sixty-nine years, and the proportion is ten males to one female. All the lesions healed spontaneously in the space of a few months.

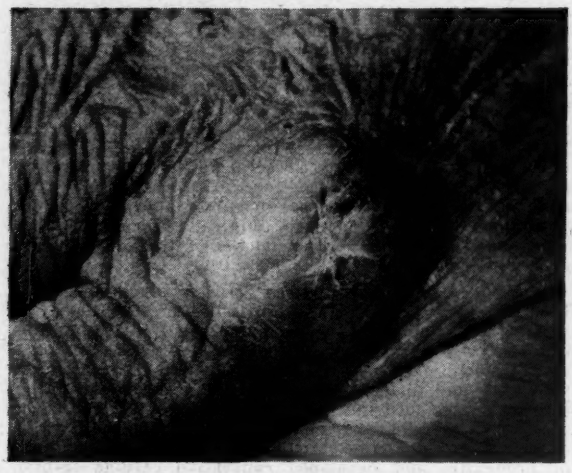

FIGURE I.

In four of the cases reported by Charteris (1951) there is a familial tendency (a father and three sons being involved), and in possibly a further two cases is there also a family history of the disease.

Macroscopically and histologically the picture in all cases appears to be identical with that seen in *molluscum sebaceum*; but the multiplicity of the lesion and its familial tendency distinguish it. An interesting feature is that there is no evidence of other precancerous lesions in the skin in spite of the multiplicity of the lesions—all develop in healthy skin.

The history of rapid growth and regression and the appearance of spread to adjacent areas seen in occasional cases suggest a virus ætiology and autoinfection. However, there is no experimental evidence that inoculation of an extract of the tumour into other skin areas is capable of inducing similar growth.

Currie and Smith (1952) record a further five cases of spontaneously healing squamous carcinoma, and reject the identity of the condition with *molluscum sebaceum*. They go further and make the following statement:

> Critical scrutiny of a large series of single squamous cell carcinomata of low grade malignancy reveals similar, though less pronounced, regressive tendencies in a surprisingly high proportion.

Tumour-Like Keratosis.

Poth (1939) describes the appearance of multiple lesions on the back of the hands in a male subject, aged forty-five years, after severe sunburn. There is a history of similar lesions having been noted previously, and in all cases the tumours healed spontaneously. The clinical and histological appearance described is essentially like that of *molluscum sebaceum*, although various pathologists reported on the same specimen as *verruca vulgaris*, squamous-cell carcinoma and tumour-like keratosis. However, multiplicity of the lesion and a background of actinic over-exposure distinguish it from *molluscum sebaceum*.

Fliegelman and Loveman (1952) report similar multiple spontaneously healing lesions on the hands of a man, aged seventy-five years, with a history of having worked with tar. Macroscopically and histologically the lesions are similar to those of *molluscum sebaceum*; but again the site and multiplicity of the lesion distinguish it.

Treatment.

Discussing the treatment of *molluscum sebaceum*, Beare (1953) advises cutting off the top of the tumour and cauterizing the base. He states that excellent cosmetic results are achieved by this method. In some cases this treatment has been followed by a single dose of X-ray therapy, 800*r* to 1200*r* being given; but Beare notes no added advantage from this treatment. A few lesions were left to heal spontaneously, and puckered scars resulted.

Rook (1950) reports a series of 40 cases; in most of them the lesions have been treated by radiotherapy. He notes that the immediate response of the lesion to radiotherapy is less brisk than that of a basal-cell or squamous-cell carcinoma of the skin. As experience developed, this author preferred simple excision or curettage, but noted spontaneous cure in only two cases of the series. This is in accordance with Beare's observation (1953) that in only four out of his series of seventy-six cases did the lesions spontaneously regress.

Finney (1954) also discusses the necessity for some form of treatment on account of the difficulty in making a certain pathological diagnosis on a biopsy specimen, and the difficulty in clinical diagnosis before the typical keratin plug has developed. He gives the results of treatment by various dosages of X-ray therapy in 25 cases. Of these patients, five had been unsuccessfully treated on a previous occasion by local excision, diathermy or the application of caustics or carbon dioxide snow. He notes that a dose of 1500*r* to 1750*r* given at one treatment (H.V.L. 3·3 millimetres of aluminium) gives satisfactory regression of the lesions in three to five weeks, with only a second-degree reaction. (This dose is about two-thirds of that required for the treatment of squamous-cell carcinoma.)

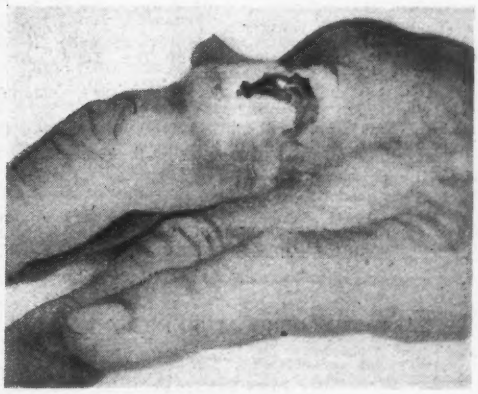

FIGURE II.

To sum up, therefore, one may theoretically leave the lesion untreated and await spontaneous regression after the lesion has reached maximum size. However, well-planned treatment, whether by surgery or radiotherapy, will lead to more rapid healing and a better scar than that following spontaneous regression, and will avoid the danger of leaving a true squamous-cell carcinoma untreated if the diagnosis is incorrect.

Report of a Case.

The patient was a male, aged sixty-six years. In 1949 a dome-shaped lesion developed on the dorsum of the right hand opposite the head of the second metacarpal bone

(Figure I). Histological examination of the lesion revealed appearances suggestive of epithelioma, and it was treated with X-ray therapy to a dose of 6000*r* in thirteen days. (On review of the sections later, the lesion was reported as *molluscum sebaceum*.)

In January, 1954, a similar lesion, 2·8 by 2·5 centimetres in area, with a keratinized centre, appeared on the dorsum of the first phalanx of the left middle finger (Figure II). A biopsy was taken, and the lesion was reported as *molluscum sebaceum* in which regression would probably occur spontaneously. In spite of this, the lesion increased rapidly in size over the following three to four months, and was therefore treated with X-ray therapy to a tumour dose of 3960*r* in five days. The lesion resolved completely after therapy, leaving a somewhat atrophic scar; but resolution was slower than that following treatment of a squamous-cell carcinoma.

Summary.

The necessity for surgical or radiotherapeutic intervention in *molluscum sebaceum* is discussed in relation to possible doubt as to diagnosis and the end results.

A case of *molluscum sebaceum* with two separate lesions on the dorsum of the hand and fingers is described. These atypical features are discussed in relation to the similar conditions of "spontaneously healing epithelioma of the skin" and "tumour-like keratosis of the skin".

Acknowledgement.

Acknowledgements are due to Dr. D. B. Pearce for permission to quote details of treatment and for a photograph of the first lesion.

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STAPHYLOCOCCAL PNEUMONIA.

By JOHN SULLIVAN,

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THIS is a discussion of eleven cases of pneumonia, the incriminating organism appearing to be the staphylococcus. All cases occurred in July, 1954. In the succeeding August no further cases occurred.

These cases followed a very similar clinical pattern. The patients were aged between seven months and three years. All had a respiratory tract infection prior to the pneumonia. This preliminary respiratory tract infection was of the nature of mild bronchitis: the patients had a loose cough, the temperature was not high, and the pertinent signs were expiratory and inspiratory rhonchi throughout the chest. The condition subsided in a few days, apparently responding to penicillin therapy. Seven to fourteen days after the bronchitis, a sudden high rise in temperature was noticed. The patients became very "mucousy", and twenty-four to forty-eight hours later respiratory distress was evident. The respirations numbered about 60 per minute. Seven of the patients incessantly tossed their heads from side to side. Two of the patients had head retraction and meningismus—this was probably caused by consolidation of the apex or apices. No abnormality was found in the cerebro-spinal fluid of these latter children. The characteristic features of their temperature charts were the sudden high rise, the sustained high plateau—a variation of only 2° F. throughout the disease—and a sudden fall to normal level about the seventh day. When the temperature fell to normal (at the "crisis"), a pronounced improvement in the patient's condition was evident. Radiologically, the appearances of the "shadows" in the early phase of the disease were those of lobar pneumonia. The patient C., who developed a pyopneumothorax, appeared to have several cavities in the lung parenchyma in the later stage of the disease. Destruction of lung tissue in staphylococcal pneumonia apparently takes place in some cases—hence the cavitation. The radiological evidence of lobar pneumonia was consistent with the clinical evidence, for clinically the disease was very similar to the lobar pneumonia of the pre-antibiotic era.

Three deaths occurred in the series; they happened in the high temperature phase, on the sixth or seventh day of disease. In four cases the temperature fell at the time expected, but not to normal. This was indicative of a complication. The patient B. continued to have signs of bronchitis and thrush of the mouth, and eleven days after the crisis developed the typical exanthem of varicella; the patient C. developed pyopneumothorax; the patient G. developed morbilli; the patient J. continued to have signs of bronchitis and developed congestive cardiac failure. These last four patients eventually recovered, and were discharged from hospital.

There is a striking consistency in the interval of onset of disease from case to case. A fresh case or cases occurred at intervals of approximately four days. It would be interesting to correlate this time factor with contact from case to case. This is possible in half the cases. For the rest, all these children except two were in the same ward for at least seven days before the onset of pneumonia. The two exceptions, patients A. and B., had sisters or brothers in this ward before they themselves developed pneumonia. It would appear that the organism had a high degree of infectivity.

Three cultures from these cases were forwarded to the Fairfax Institute of Pathology, Royal Prince Alfred Hospital, Sydney, and a report was obtained. It read as follows:

> All three staphylococcal cultures are resistant to penicillin, streptomycin, aureomycin, but sensitive to chloramphenicol. Bonegilla 2 and E.T. are lysed by phages 47, 53, and 77 applied undiluted. I think, however, that all three are identical but that Bonegilla 3 is partly phage resistant. This phage pattern puts the strains in Phage Group III and it is interesting that nearly all the antibiotic resistant strains that we have isolated from cross infected cases belong to this group. It is almost certain therefore that the strains were acquired by these children while in hospital.

It would appear from Table II that treatment was not given in accordance with laboratory findings. The explanation was that the endemic was well under way before its nature was realized, and swabs were taken mainly in the later stages of the disease; swabs had to be sent to an outside laboratory, and then there was the time lag of the growth and sensitivity tests of the culture. On the pathological findings it would appear that the incriminating organism was sensitive mainly to "Chloromycetin" and "Terramycin" (see patients B., C. and D. in relation to "Chloromycetin", and patients C., F., G. and K. in relation to "Terramycin"). Patient B. received "Chloromycetin" and "Terramycin" without any apparent effect; patient C. received "Terramycin" without apparent effect, and further-

TABLE I.

Patient.	Age in Months.	Recent Prior History.	Temperature at Onset.	Result.	Chemotherapy.
A	36	Cough for 7 days.	104° F. on July 3.	Death on July 10.	*Penicillin, July 4 to 6.* "Sulphatriad", July 5 to 10. Streptomycin, July 7 to 10.
B	9	Bronchitis on June 21.	103·5° F. on July 3.	Rapid fall of temperature on July 13; dyspnœic for weeks after.	Penicillin, June 29 to July 4. "Chloromycetin", July 4 to 6. Streptomycin, July 7 to 9. *"Terramycin", July 10 to 23.*
C	12	Bronchitis on June 30.	104° F. on July 8.	Rapid fall of temperature on July 15, *then pyopneumothorax.* Recovery.	Streptomycin, July 8 to 11. "Terramycin", July 11 to 18. "Erythrocin", July 22 to August 7.
D	18	Bronchitis on July 2.	104·6° F. on July 12.	Death on July 19.	Penicillin, July 9 to 12. "Terramycin", July 14 to 19.
E	24	Morbilli on July 5.	105·8° F. on July 13.	Death on July 19.	Penicillin, July 6 to 13. "Terramycin", July 13 to 19.
F	14	Bronchitis on July 1.	106·6° F. on July 16.	Rapid fall of temperature on July 22; dyspnœic for weeks.	"Chloromycetin", July 15 to 19. Streptomycin, July 19 to 21. Penicillin, July 19 to 21. "Ilotycin", July 21 to August 2.
G	8	Bronchitis on July 11.	104·8° F. on July 16.	Rapid fall of temperature on July 24; dyspnœic for weeks.	Penicillin, July 12 to 21. "Erythrocin", July 22 to 31.
H	8	Bronchitis on July 5.	103·6° F. on July 20.	Rapid fall of temperature on July 27; complete recovery.	Penicillin, July 20 to 24. "Sulphatriad", July 20 to 24. "Erythrocin", July 24 to August 2.
I	7	Bronchitis on July 13.	102·4° F. on July 21.	Rapid fall of temperature on July 27; complete recovery.	*Penicillin, July 14 to 23.* "Erythrocin", July 23 to August 4.
J	8	Upper respiratory tract infection on July 2.	104·4° F. on July 23.	Rapid fall of temperature on July 29; congestive cardiac failure; recovery.	"Erythrocin", July 23 to August 9.
K	24	Morbilli on July 19.	105·4° F. on July 27.	Rapid fall of temperature on August 1; complete recovery.	Penicillin, July 26 to 29. "Sulphatriad", July 26 to 29. "Chloromycetin", July 29 to August 4.

more the sensitivity of the staphylococcus had apparently "changed" between July 20 and 27; patient F. received "Chloromycetin" from July 15 to 19 without effect. Therefore, if one presumes that the same strain of staphylococcus was implicated throughout the series, as seems reasonable, then the in-vivo response was different from that expected from the results of in-vitro sensitivity tests.

Treatment.

In treatment penicillin, penicillin and "Sulphatriad", "Chloromycetin", "Terramycin", streptomycin and streptomycin with penicillin were tried unsuccessfully. Therefore, as the foregoing agents did not appear to affect the course of the disease, erythromycin and staphylococcal antitoxin were used in conjunction in the later cases. One hundred milligrammes of "Erythrocin" were given orally every four hours, and 2500 units of antitoxin per day. Despite these measures the disease appeared to run its course. However, there were no deaths among the patients who had this treatment, and their convalescence appeared to run more smoothly than that of the earlier patients.

As supportive measures, frequent, small feedings were given. A fluid intake-output chart was kept. Oxygen was given for ten to twenty minutes per hour in the more distressing phase of the disease.

It is suggested that in pneumonia the steam tent should not be used. Before the true nature of the disease was realized and because of the "croupy" respirations of one

TABLE II.

Patient.	Pathological Findings.	Sensitivity.	Chemotherapy.
B	July 12: Swab of fauces: staphylococci.	Not tested.	Penicillin, June 29 to July 4. "Chloromycetin", July 4 to 6. Streptomycin, July 7 to 9. "Terramycin", July 10 to 23.
C	July 12: Swab of fauces: staphylococci and α-hæmolytic streptococci. July 20: Hæmolytic staphylococcus. July 27: *Staph. aureus.*	Not tested. To "Chloromycetin". To "Aureomycin", "Terramycin" and streptomycin.	Streptomycin, July 8 to 11. "Terramycin", July 11 to 18. "Erythrocin", July 22 to August 7.
D	July 19: Smears from larynx *post mortem*: staphylococci.	To "Chloromycetin" (see below).	Penicillin, July 9 to 12. "Terramycin", July 14 to 19.
F	July 20: Swab of fauces: *Staph. aureus.*	To "Aureomycin", "Chloromycetin", "Terramycin" and streptomycin.	"Chloromycetin", July 15 to 19. Streptomycin, July 19 to 21. Penicillin, July 19 to 21. "Ilotycin", July 21 to August 2.
G	July 29: Swab of fauces: *Staph. aureus.*	To "Terramycin" and streptomycin.	Penicillin, July 20 to 24. "Erythrocin", July 22 to 31.
K	July 28: Swab of fauces: *Staph. aureus.*	To "Aureomycin", "Terramycin" and streptomycin.	Penicillin, July 26 to 29. "Sulphatriad", July 26 to 29. "Chloromycetin", July 29 to August 4.

of the early patients, one small patient was put in a steamed room. This was a small room with a heavy atmosphere of steam. The impression was that the child appeared to be eased. A second patient, whose distress appeared to be attributable to excess mucus in the respiratory tract, also appeared to be benefited. This is merely a clinical impression.

The child who developed pyopneumothorax had daily aspirations of thick yellow pus. Up to 60 cubic centimetres were aspirated. Dornase was injected into the affected pleural space to facilitate the procedure. At the end of ten days the empyema did not appear to be lessening. Therefore rib resection was performed, drainage was implemented and the patient made good progress.

Post-Mortem Examination.

The following are the post-mortem findings on patient D., a male child, aged eighteen months.

The body was that of a well-nourished infant. When the chest was opened, no fluid was found in the pleural cavity on either side. The right side of the heart was greatly dilated, and on superficial examination the lungs presented the appearance of severe inflammation; they were deep purplish in colour with scattered areas suggesting consolidation. Some scattered areas of consolidated lung tissue about one and a half inches in diameter could be felt throughout both lungs. The cut surface of the lungs exuded pus from cut bronchi throughout the whole of both lungs. Consolidated areas having a lobular distribution were present throughout both lungs. The trachea and main bronchi were removed and opened. They were almost completely obstructed by thick yellow mucopus, of which cultures and smears were made. There was no fluid in the pericardial sac. The right auricle was greatly distended and filled with post-mortem clot. The valves appeared normal. The heart muscle was friable and appeared to be affected by toxic degeneration. The liver was pale, fatty and friable. The spleen was enlarged to the size of a normal adult spleen, with rounded edges and a pronounced splenic notch. It was a dark purple colour and felt hard. Apart from some congestion the kidneys appeared to be normal. The suprarenal glands were normal, as were also the stomach and intestines.

Histological examination of sections of the trachea and lungs showed in the former acute purulent tracheitis with total destruction of the mucosa and submucosa. Numerous staphylococci were seen throughout the section. A pure culture of *Staph. aureus* was obtained from the exudate; this has since been found to be completely resistant to penicillin, streptomycin, "Terramycin" and "Aureomycin". It was sensitive to "Chloromycetin". The cause of death was recorded as acute staphylococcal pneumonia, probably secondary to influenza, with terminal toxæmia.

Summary.

Eleven cases of staphylococcal pneumonia occurring in young children are recorded.

There were three deaths. Another patient developed pyopneumothorax, but recovered. Three further patients had mild dyspnœa three weeks after remission of the disease.

In vivo, the incriminating organism was resistant to the known antibiotics.

Acknowledgements.

My thanks are expressed to the Director-General of Health for permission to publish this article, and also to Dr. J. L. O'Connor, of the Commonwealth Serum Laboratories, to Dr. Phyllis Rountree, of the Fairfax Institute of Pathology, to Dr. H. Hambrett, of the Commonwealth Health Laboratory, Albury, and to Dr. H. Franklands, Deputy Director of Health, Victoria, for their help and advice.

THE SPOT-MAP IN EPIDEMIOLOGY: A NOTE ON THE USE OF SPOT-MAPS DURING TWO EPIDEMICS OF POLIOMYELITIS IN WESTERN AUSTRALIA.

By D. J. R. Snow,
Epidemiologist, Department of Public Health, Perth.

The use of the spot-map is one of the simplest techniques available in the study of epidemics. It was probably used first by John Snow in 1854, during his classical investigation of the cholera epidemic associated with the Broad Street Pump. His spot-map, which showed the distribution of cholera cases in relation to the wells in the district, led to an elementary deduction which has had a profound influence on disease prevention for a hundred years.

Since that time the spot-map has been used repeatedly for the purpose of identifying the sources of infection in explosive outbreaks of disease. Occasionally it has been used to determine the pattern of spread rather than the source of infection. Its value in different situations varies greatly, but in certain circumstances it still provides a helpful tool in epidemiological investigation, particularly in the study of poliomyelitis.

It is the purpose of the present note to illustrate its use in two epidemics of poliomyelitis in Western Australia, and to describe a simple modification in technique whereby the "march" of spread of the disease can be studied more readily by means of serial spot-maps.

If consecutive notifications throughout an entire epidemic are plotted on a single map, individual or isolated features are soon obscured in a general pattern of limited significance. If, however, the progress of an epidemic is recorded on a succession of spot-maps, more useful information is likely to be revealed.

Western Australia has experienced two considerable epidemics of poliomyelitis in the last eight years. The first involved 311 cases in 1948, and the second 436 cases in 1954. In both, rather more than half the total number of notifications were recorded from the capital city of Perth, and a series of spot-maps was prepared for this urban area.

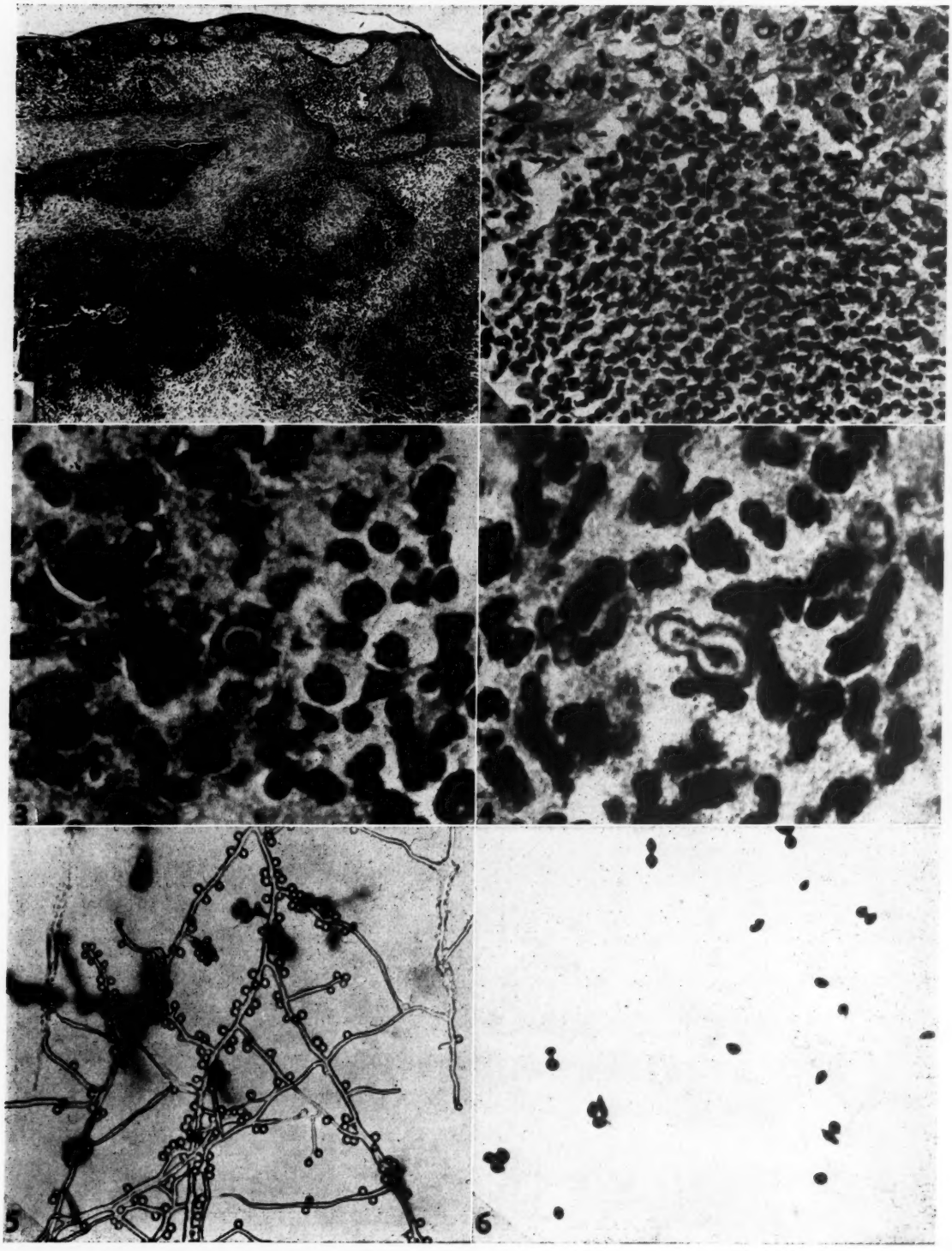
These maps (Figure I) were constructed by plotting notifications in progressive sequence according to the formula $a + 2n$, where a is an arbitrary number (such as 10) and n the preceding number in the series. In such a series where $a = 10$, the sequence would be 10, $10 + (2 \times 10)$ or 30, $10 + (2 \times 30)$ or 70, and $10 + (2 \times 70)$ or 150. In other words, the first spot-map showed the first 10 cases in the epidemic, the second map showed the first 30 cases (including the first 10), the third 70 cases (including the first 30) and so on until the total number of recorded cases in the area was exhausted. In this way a comparative pattern was obtained which revealed information not readily available by other means.

The conjoined area of Perth-Fremantle is conveniently divided by the Swan River into northern and southern parts. The 1948 epidemic appeared to commence north of the river, progressed rapidly in that sector and maintained its preponderance there throughout; further, a few fairly well-defined clusters were noticeable on the spot-maps. The southern sector was involved very late in the epidemic, and then only sparsely. The direction of spread was not specially selective, and incidence appeared to be related to population density.

The 1954 epidemic, on the other hand, seemed to develop south of the river and spread fast in that sector. However, the northern districts soon became involved, but were not as heavily affected as they had been in 1948. Again, occasional groups of cases were discernible at various stages, but were eventually obscured as more and more were plotted. Finally, no selective pathway was conspicuous.

The general impression received from a study of these spot-maps was that the 1954 epidemic developed in and mainly involved that region which was least affected in

ILLUSTRATIONS TO THE ARTICLE BY C. C. J. MINTY, D.T.R., M.C.R.A., M. MEAD AND M. F. McCAFFREY.



ILLUSTRATIONS TO THE ARTICLE BY RICHARD FLYNN AND R. TINNING.

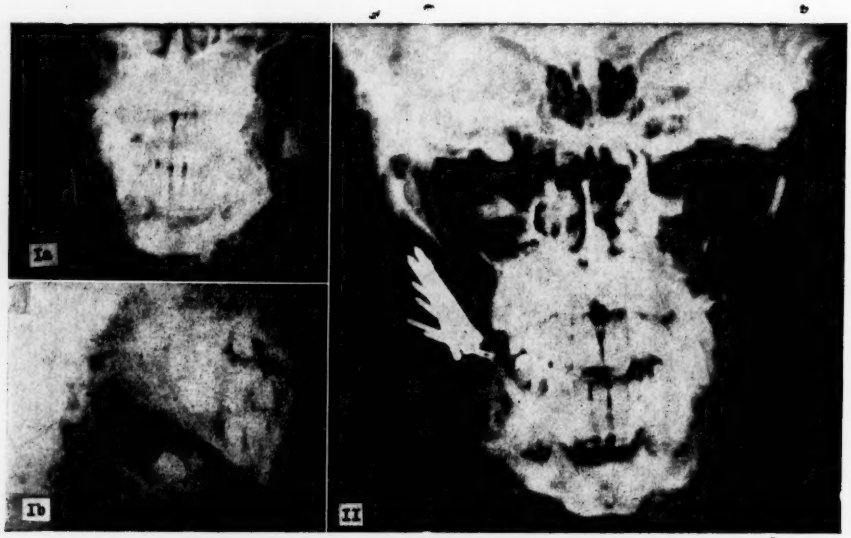


FIGURE III.

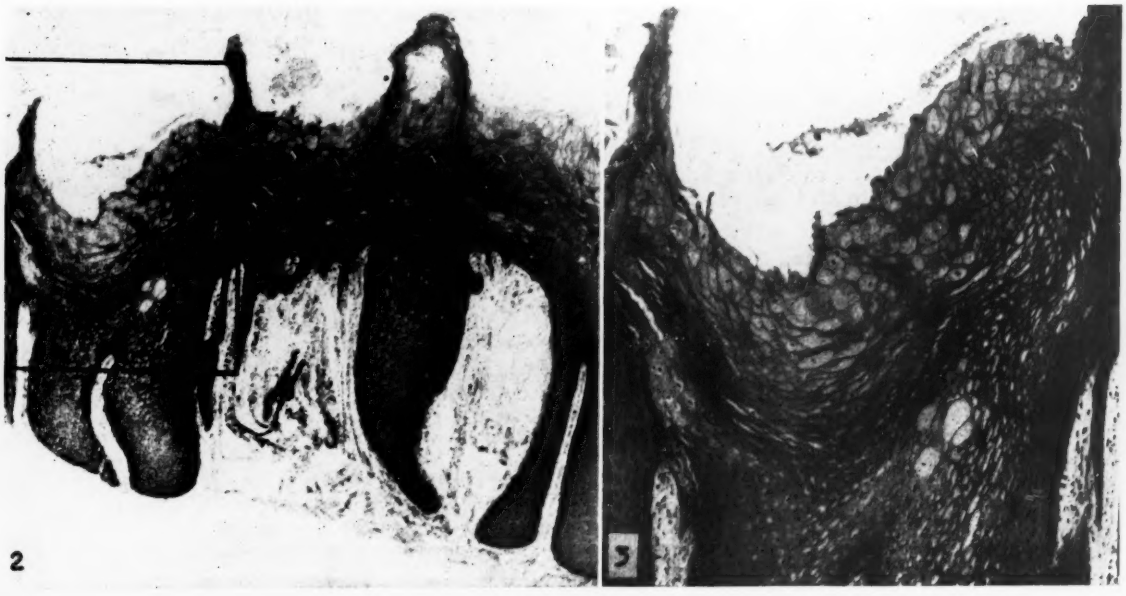
FIGURE V.

FIGURE VI.

ILLUSTRATION TO THE ARTICLE BY P. W. GILL AND J. S. JONAS.

FIGURE I.

ILLUSTRATIONS TO THE ARTICLE BY L. G. ABBOTT AND L. A. MUSSO.



ILLUSTRATIONS TO THE CLINICO-PATHOLOGICAL CONFERENCE.

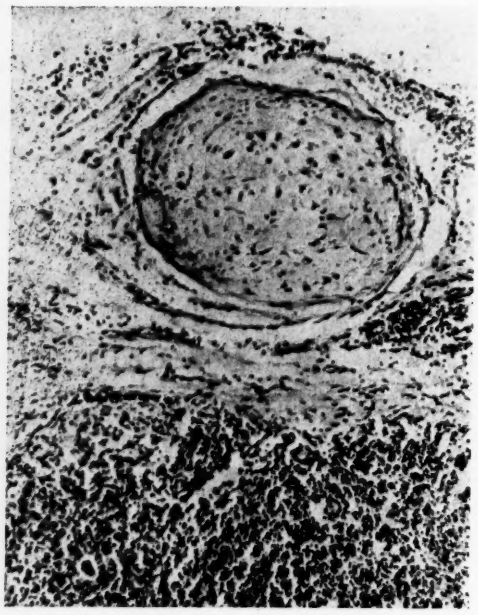


FIGURE I.

An oval deposit of growth is present in a lymph vessel in the capsule of the lymph node.

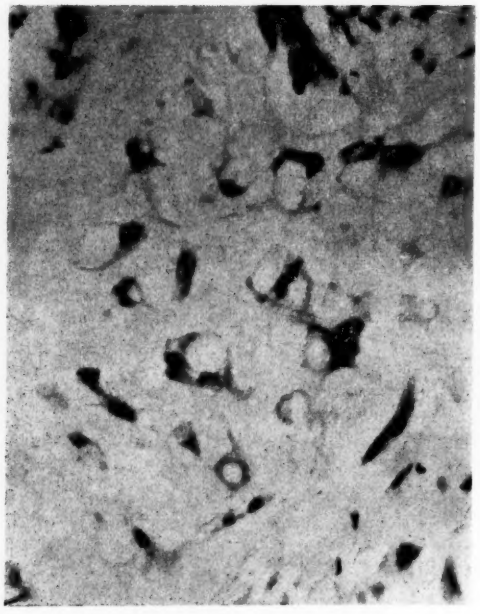


FIGURE II.

Field from a subpleural deposit of growth showing cells with large vacuoles in the cytoplasm. Some of these cells are multinucleated. The background was pale and hyaline.

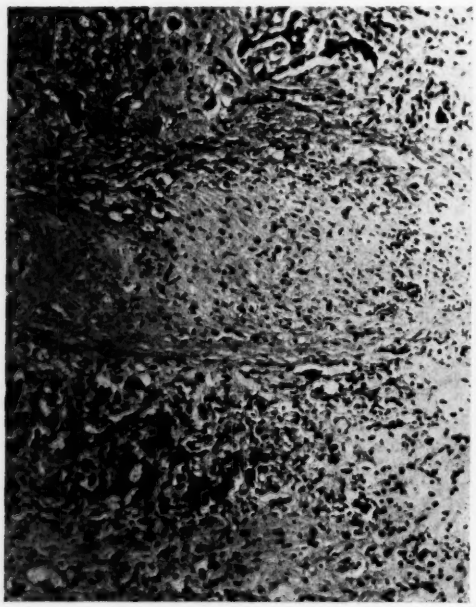


FIGURE III.

Liver showing much replacement with fibro-cellular tissue. At least part of this tissue is neoplastic. The central horizontal band in this photograph is probably tumour within a branch of the portal vein.

FIGURE IV.

Liver showing increase in fibro-cellular tissue which may be in part neoplastic. A crescentic group of cells with hyperchromatic nuclei projects into the lumen of a small branch of the portal vein. These cells are presumably neoplastic.

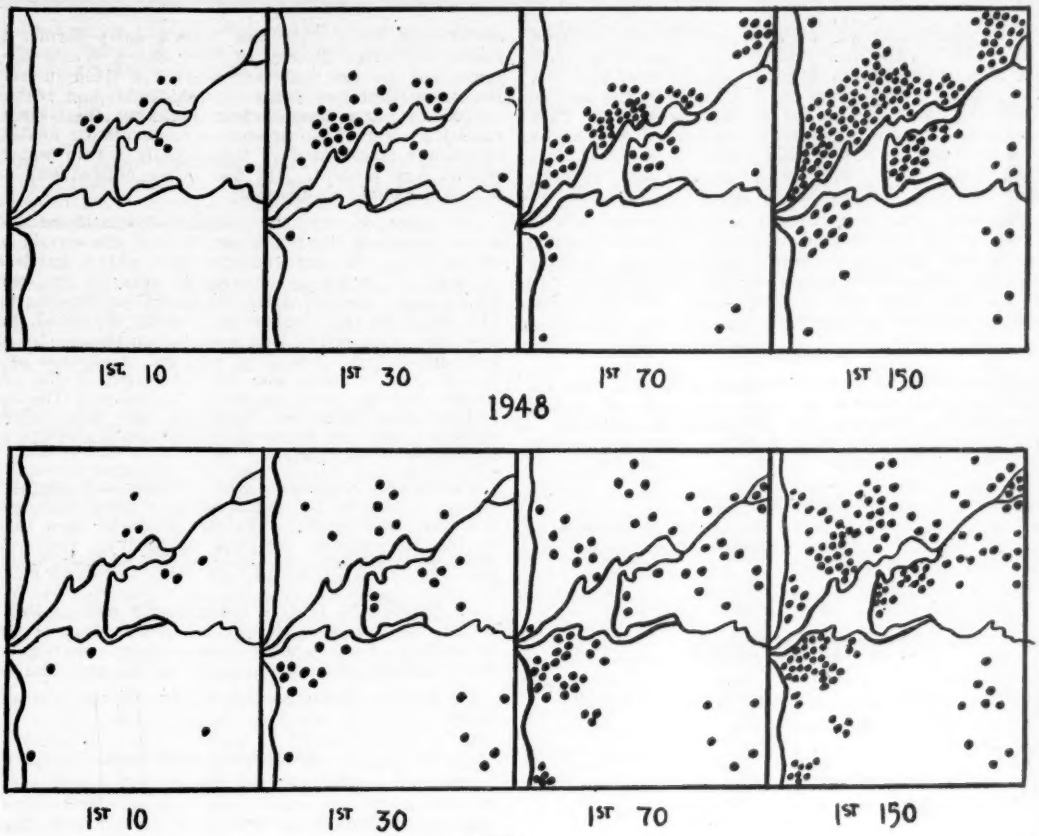


FIGURE I.

Serial spot maps of the two poliomyelitis epidemics in Perth, Western Australia.

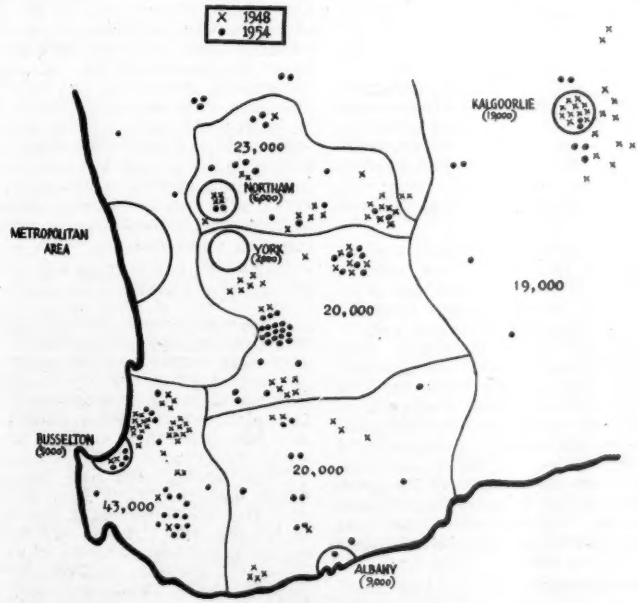


FIGURE II.

Serial spot map of the two poliomyelitis epidemics in Western Australia (excluding Perth).

1948, while that which was worse affected in 1948 suffered least in 1954. This observation is of special interest because it is in accordance with the fundamental principle that the pattern of an epidemic is influenced by the pattern of preexisting immunity to the disease concerned. Thus the substantial immunity persisting in those areas heavily involved in 1948 gave appreciable protection in 1954.

Figure II is a map showing that portion of the State of Western Australia (excluding the Perth area) affected by both epidemics. The main country centres are shown with their populations and the populations of the districts which they serve, and the occurrence of individual cases of poliomyelitis has been plotted separately for each epidemic. In general, those areas heavily seeded in 1948 were less affected in 1954 and vice versa.

Summary.

A new technique of serial spot-maps devised during the study of epidemic poliomyelitis is described. It is suggested that this technique can be applied to epidemics of other diseases and may furnish information not readily available by other means.

Acknowledgement.

I am indebted to Dr. Linley Henzell, Commissioner of Public Health for Western Australia, with whose permission this note is published.

Reports of Cases.

SPOROTRICHOSIS: A CASE REPORT FROM QUEENSLAND.

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and

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Sporotrichosis is a rare disease in Australia, only two cases having been reported previously (Robinson and Orban, 1951; Barrack and Powell, 1952). The infection in both these cases was apparently acquired in northern New South Wales, although in one (Barrack and Powell, 1952) the diagnosis was made in Queensland. The condition appears to be common in South Africa and not rare in America and Europe.

The causal organism is *Sporotrichum schenckii*, a plant saprophyte which, however, is pathogenic for several animals, including man. Infection occurs at the site of an injury to the skin. The initial lesion appears after an interval of from seven days to three months, and usually takes the form of a red, indurated, painless papule. This is not accompanied by any constitutional disturbance. Sinuses or open ulcers may develop and discharge for long periods. Later, nodules may appear along the path of the lymph vessels draining the area and may ulcerate. The regional lymph nodes are not usually enlarged, and involvement of tissues other than the skin and subcutaneous tissue is rare, although involvement of the lungs and other viscera has been described. Rarely, disseminated ulcerated lesions develop, and hypertrophic forms may result from the formation of exuberant granulation tissue.

Clinical Record.

A, a retired dairy farmer and bank messenger, aged seventy-two years, attended the Queensland Radium Institute on September 23, 1954, complaining of an ulcer on his left wrist. He had been a dairy farmer in the Richmond River district of New South Wales for forty years, but for the past four years had lived in Brisbane. He stated that two years previously he had injured his left wrist on a wooden box, breaking the skin. The resulting ulcer at times seemed to be almost healed, only to become larger again. Occasionally a thin watery discharge was present. He had never noticed any similar lesion elsewhere on his body.

The lesion of which he complained was situated on the dorsal aspect of the left wrist, close to the styloid process of the ulna. It consisted of a pale, shiny, hairless area of skin, 5·0 by 2·5 centimetres in area, in the centre of which was a shallow ulcer, 2·0 by 2·5 centimetres in area. The edges of the ulcer were slightly thickened, and the base was composed of thin granulation tissue. No sloughs were present, but the ulcer was partly covered by a dry brown crust. There was no induration of the adjacent tissues and no discharge from the lesion. The regional lymph nodes were not enlarged, nor were there any nodules along the course of the lymphatics of the arm or elsewhere on the body.

Portion of the lesion was excised, and sporotrichosis was suggested by the pathologist. Culture was attempted but was unsuccessful. Further material was then sent to the Queensland Institute of Medical Research, and *Sporotrichum schenckii* was grown in cultures made from it.

On October 26, 1954, a radon mould was applied to the lesion, calculated to deliver a surface dose of 4000*r* in seven days ($h = 0{\cdot}5$ centimetre). An erythematous reaction was produced, and on December 25 the area had healed.

No further manifestation of the disease has appeared to date.

Pathological Histology.

The tissue submitted for examination was a small piece of skin about one centimetre long, one millimetre broad and two millimetres in depth. No subcutaneous tissue was present. The specimen was received fixed in 10% formalin.

In sections cut from paraffin blocks and stained with hæmatoxylin and eosin, the general appearance was that of a granuloma with a moderate amount of epithelial hyperplasia (Figure I). In the dermis there was evidence of œdema and increased vascularity. The most prominent features were epidermal and dermal abscesses containing many polymorphonuclear leucocytes, a few macrophages, eosinophile leucocytes and cellular debris. The dermal abscesses were surrounded by a broad but ill-defined layer of epithelioid cells and a few foreign-body giant cells (Figure II). Outside this layer there was a diffuse cellular infiltration composed of macrophages, plasma cells, lymphocytes, eosinophile and neutrophile leucocytes, foreign-body giant cells and fibroblasts. Mast cells were not plentiful in this case, although their presence is stressed in many reported cases, nor was there a distinct "syphiloid zone".

Although no organisms had been found at this stage, the diagnosis of sporotrichosis was suggested.

A careful search through serial sections stained with hæmatoxylin and eosin was then made, but fungal cells were very scanty. In fact, only two yeast-like forms, each apparently lying free in an abscess, were found. These cells could not be distinguished under the low-power objective, although they were readily visible at a magnification of × 360. One fungal cell had an oval outline and measured 5μ by 6μ (Figure III). Its prominent refractile cell wall stained faintly pink and was 0·6μ thick. Inside this a broad unstained band gave a false impression of capsulation and was apparently due to shrinkage (Kligman and Baldridge, 1951). The central mass of shrunken protoplasm measuring 2μ by 3μ had a granular appearance with a few clear areas resembling vacuoles, and was a distinctive pale brownish-blue colour. A thin, irregular layer of eosinophilic material was present on the outer aspect of the cell wall, giving it a tuberculate or spiny appearance. This layer resembled the encrustation often found on

pathogenic fungi in tissues (Moore, 1946), but no radiate formations or "asteroid bodies", described as being diagnostic of sporotrichosis, were found.

The other fungal cell found was similar in appearance, but slightly larger in size, and exhibited budding (Figure IV).

No fungi or other organisms were found in sections stained by Gram's method, or in the small amount of material available for staining by the Hotchkiss-McManus method (periodic-acid Schiff stain).

Mycology.

Biopsy material was teased out aseptically, inoculated onto Sabouraud's glucose agar and Littman's medium, and incubated at 28° C. and at 35° C. Microscopic examination of teased material mounted direct in lactophenol and in potassium hydroxide showed no evidence of pathogenic fungi.

The fungus was first visible on Sabouraud's medium at 28° C. six days after inoculation. At first the colonies were small, cream-coloured and velvety, but later they became moist, wrinkled in the centre, and gradually deepened in colour until they were a dark brown. The fungus also grew on Littman's medium at 28° C. and on Sabouraud's medium at 35° C., but more slowly.

The morphology of the organism was studied from slide cultures on corn-meal agar. Microscopically the colony was composed of fine, septate, branched hyphæ, measuring 1·5μ to 2·0μ in diameter, and bearing oval to pyriform conidia singly or in clusters. The conidia measured 2·5μ to 3·0μ by 1·5μ, and were borne either terminally or laterally on short branches or on the main hyphæ (Figure V).

A yeast-like phase was obtained on glucose-cystine-blood agar at 35° C. Soft greyish colonies developed after transfer from Sabouraud cultures. These colonies consisted mainly of elliptical, budding, yeast-like cells measuring 1·5μ to 2·5μ by 2·5μ to 4·0μ, with a few pieces of mycelium (Figure VI).

The behaviour of the organism on beef extract agar, potato dextrose agar, malt agar, corn-meal agar, Czapek's medium, and the chorio-allantoic membrane of embryonated eggs, and over a range of temperatures from 10° C. to 45° C., was identical with that of the organism isolated by Barrack and Powell (1952).

The fungus was therefore classified as *Sporotrichum schenckii* var. *beurmanni* (Matruchot and Ramond, 1905) Dodge, 1953.

Discussion.

No previous case of sporotrichosis has been described as originating in Queensland, and the fact that only two other cases are recorded in the Australian literature indicates that the disease is rarely diagnosed. However, it is not rare in other parts of the world, and it was reported in 1947 that 2825 cases had occurred on mines of the Witwatersrand. It is suggested that the disease may not be rare in Australia, and that it remains undiagnosed because of one or more of three factors. Firstly, the organisms are often scanty, and may be missed in a routine histological examination, unless serial sections are examined. Secondly, the bacterial contamination, which readily occurs in a discharging lesion, may mask the presence of the fungus when culture is attempted. Thirdly, the clinical appearance, as illustrated by the case reported, may differ greatly from classical descriptions of the disease.

With regard to treatment, the radiotherapeutic approach was suggested by the apparent localization of a lesion which had existed for two years without lymphatic spread. It therefore seemed reasonable to adopt a local method of treatment, which experience had proved to be effective in another variety of localized fungus infection (chromoblastomycosis). Various authors (Sievers, 1954; Mackee and Cipollaro, 1946) have mentioned the usefulness of radiotherapy. It is recognized that for more widespread forms of the disease the treatment of choice is the exhibition of iodides. Stilbamidine has been recommended for those patients not benefited by iodine therapy (Harrell, Bocobo and Curtis, 1954). However, the response to treatment in the case described was satisfactory, and more rapid than might have been expected from iodides alone.

Summary.

A case of sporotrichosis arising in Queensland is described, with an account of the pathological and cultural findings.

Acknowledgements.

We are indebted to Dr. M. J. Mackerras, of the Queensland Institute of Medical Research, for photographing the fungus and the sections, and to Mr. R. Findlayson, of the University of Queensland Medical School, for the Hotchkiss-McManus stain.

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Legends to Illustrations.

FIGURE I.—Photomicrograph of section showing epithelial hyperplasia and a small thick-walled abscess in dermis (×40).

FIGURE II.—Photomicrograph showing abscess and surrounding zone of epithelioid cells (×470).

FIGURE III.—Single fungal cell showing eosinophilic encrustation (×1850).

FIGURE IV.—Fungal cell exhibiting budding (×1850).

FIGURE V.—Slide culture of *Sporotrichum schenckii*, showing hyphæ and conidia (×470, unstained).

FIGURE VI.—Yeast phase of *Sporotrichum schenckii* (×1100, Gram stain).

AN ADAMANTINOMA IN A DENTIGEROUS CYST.

By RICHARD FLYNN AND R. TINNING,
Sydney.

Clinical Record.

A FEMALE patient, aged nineteen years, referred by Dr. E. Stokes, consulted me in September, 1935, on account of a swelling of the right side of her face and mouth. She had consulted her dentist in her home town twelve months prior to her coming to Sydney; he had explored the swelling intraorally. From the site of this exploration a fleshy mass now protruded. She complained of this, and also of swelling and discharge into her mouth. The swelling was painful only when the discharge was blocked and the swelling increased in size. She also stated that the right lower wisdom tooth had not "come through".

On examination of the patient an ovoid swelling was palpated; it was about the size of a pullet's egg (five by three centimetres in area) and involved the angle of the mandible on the right side and most of the ascending

ramus. On inspection of her mouth the size of the swelling was confirmed, also the absence of her right lower wisdom tooth. A fleshy mass protruded from the site of the exploration. No swellings were noticed in her other bones or complained of. A radiograph at this time showed a large cystic area occupying and expanding the angle and the ascending ramus of the mandible. The radiologist considered the lesion to be a dentigerous cyst (Figures Ia and Ib).

On October 4, 1935, through a skin incision at the angle of the mandible, the ascending ramus of the mandible was explored and the lateral wall of the cyst was exposed and a hole made with a burr; the wall was extremely thin. The wall of the cyst was opened and the glittering fluid contents were sucked out, and the membrane lining the cyst along with its fleshy contents was curetted out. The right lower third molar tooth was also removed. Some of the fleshy contents along with a piece of the lining membrane were sent for pathological examination. The cyst

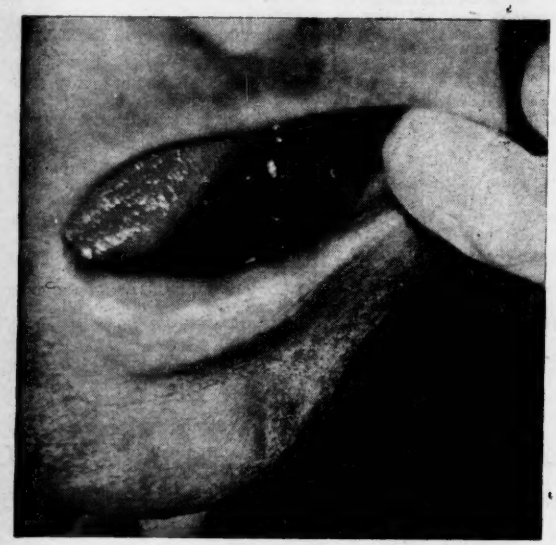

Figure IV.

cavity was then packed with gauze soaked in adrenaline, and a piece of this was pulled into the mouth through the hole which had been produced in the dental operation. The incision was then closed in layers. The pathological report was as follows: "Sections show œdematous connective tissue and some stratified epithelium. No evidence of malignancy."

Because of my interest in the condition, and knowing Dr. G. Davies's interest, I took some of the sections along to him and he gave me the following report:

> The lining of the cyst submitted is formed of epithelium which has a papillary structure. The cores of the papillæ are œdematous and apparently were highly complex. The epithelium covering the papillæ forms a reticulated structure. In some areas it resembles stratified squamous epithelium except in that the cells are slightly more separated from each other than usual and that they have a slightly stellate shape. In other parts this separation is much more marked and the stellate shape is much more pronounced so that the epithelium resembles that of an adamantinoma. Many sections have been taken and in the majority the epithelium is of this nature. In a few small areas it is more smooth and is seen as a thin layer of stratified epithelium. Dentigerous cyst lined by highly papillary epithelium which resembles that of an adamantinoma.

On two occasions the packing was removed and the cyst was repacked with plain gauze before the patient was discharged from hospital with the neck incision healed. The patient reported back on February 6, 1936. The swelling was smaller and there was nothing protruding through the hole in the mouth. But when she reported again on June 7, 1936, for a follow-up examination, though the swelling of her face was less, from the opening into her mouth some soft tissue was projecting. Accordingly, on June 8, 1936, under regional anæsthesia the cyst was curetted and radium needles were inserted (five two-milligramme needles and four one-milligramme needles) through the hole in the ascending ramus (Figure II). Dr. C. H. Shearman reported as follows on the tissue obtained by curettage:

> Microscopic section shows epithelial hyperplasia with connective tissue stroma which varies in appearance in different parts of the section. Some appear to be undergoing degenerative changes. The epithelium is basal cell in type and occurs in irregular strands though some larger groups of cells are seen. The condition is one of epithelial odontome (Adamantinoma).

Dr. E. Thomson, who was relieving Dr. G. Davies, reported as follows on slides: "Sections show the structure of an adamantinoma."

The radium needles were left in for twelve hours, the total dose thus being 1288 milligramme-hours. I was afraid to leave them longer on account of the risk of bone necrosis. No redness of the skin of the face developed, though some redness was seen around the opening of the sinus into the mouth. The patient was kept under observation. On April 14, 1937, I made a note that the small hole in the mouth was covered by mucous membrane, and on November 28, 1938, I wrote that the bone felt normal in size, the hole in the mouth was healed, and the facial asymmetry had gone.

During the following years the patient was busy rearing her family, and I did not see her again till July 2, 1954, when she returned at my request. I then noted that her face was quite symmetrical, that the scar had migrated down her neck a little but was practically invisible, and that the hole in her mouth had remained healed. I referred her to Dr. F. J. McEncroe for further radiographic examination because of his interest and experience in jaw pathology, and he reported as follows on the radiographs (Figure III):

> There is slight generalized bony irregularity on the right side of the mandible in the neighbourhood of the angle the result of old surgical procedures. No significant evidence of osseous lesion is now apparent in this region and the rest of the mandible appears normal.

Comment.

The case is reported because it shows the effect of radium on an adamantinoma occurring in a single unilocular cyst. At the same time it must be emphasized that radium has no place in the treatment of multilocular cysts any more than simple curettage has. The treatment of these tumours is wide resection and, to prevent deformity, bone grafting. The bone graft can be put in at the first operation if the patient's condition is good; otherwise it can wait till the patient has recovered from the effects of excision of the jaw. "Widely" must be emphasized, because recurrences of these tumours are not really recurrences, but further growth of cysts already present at the time of the original operation. Also I think it is worth emphasizing that there is no need for the use of bone chips in dealing with large cysts, as when the lining membrane has been removed the bone around the cyst regenerates, and within twelve months it is difficult even by X-ray examination to determine the side of the cyst. The use of bone chips only adds a further possibilty of complication, and in this case it was definitely contraindicated owing to the cyst's having been exposed to infection from the mouth for a long time.

I have treated a second patient suffering from an adamantinoma in a follicular cyst (Figures IV and V). The lesion was cured by radon needles, and the patient's mandible was reorganized till at a later stage it was impossible to determine the site of the cyst. The X-ray film (Figure VI) showed a picture similar to a healthy

edentulous jaw, and it was liable to the same risk of fracture; this was proved by the fact that a bump from the patient's child's head caused a fracture which united after surgical treatment.

FULL-TERM PREGNANCY AND CONFINEMENT OF A PRIMIPARA WITH TRIPLETS.

By P. W. GILL AND J. S. JONAS,
Singleton, New South Wales.

Clinical Record.

G.P., A PRIMIPARA, aged nineteen years, of Australian parentage, was born in Singleton. She was a strong, healthy young woman of medium build and happy disposition. Her last menstrual period, which was normal, commenced on April 29, 1953. The expected date of delivery was February 11, 1954.

Her first consultation was on August 10, 1953. She was then thirteen weeks pregnant (by dates), but the fundus was at the eighteen-weeks level. She had had no sickness, but came because of urinary tract infection, which responded promptly to the exhibition of sulphonamides and alkalis. Fœtal movements were first noted on September 23, when she was nineteen weeks pregnant (by dates) and the fundus was at the twenty-eight-weeks level. The uterus was unusually wide, and numerous fœtal parts were felt. X-ray examination was carried out on November 13 by Dr. T. W. Anderson. This revealed triplets, all three presenting by the breech (Figure I).

At this stage the onset of intermittent hæmorrhage from the vagina (bright blood) caused some anxiety. It occurred mainly if the patient sat in a hot bath or stooped. Three breech presentations and the symptomless loss of bright blood at twenty-eight weeks conjured up the spectre of *placenta prævia*. However, we were fortified by reassurance from a senior colleague, and the threat did not materialize. Bleeding recurred for about a month, then ceased and did not occur again. (Subsequently, moreover, each fœtus was delivered by the vertex.)

The patient was admitted to hospital on December 26 (at nearly thirty-three weeks) because of excessive gain in weight, rising diastolic blood pressure and commencing œdema of the lower limbs. There was no albuminuria until two weeks later. She remained in hospital till term, and the benefit of this is seen in the chart (Figure II). Throughout her stay in hospital she was permitted to go to the bathroom each day, but otherwise remained in bed. A generous protein intake was allowed; salt, fats and carbohydrates were restricted. Half a grain of phenobarbital was given three times a day.

Albuminuria appeared first on January 11. It appeared intermittently for the first week, and was never absent after Januarly 18. It varied from a light cloud to a heavy cloud without deposit until January 30, when there was a "one-twelfth" deposit. It did not exceed this amount until the afternoon of February 3, when "one-quarter" of albumin was present and the patient was uncomfortable. Labour was thought to be imminent, and she was given an intramuscular injection of ten milligrammes of "Synkavit" to boost the fœtal prothrombin levels. As a preventive against intrapartum nausea and cramps she also received by intravenous injection one cubic centimetre of "Pyridoxine Compound" (Fawns and McAllan) and ten cubic centimetres of 10% calcium gluconate solution. Three grains of "Nembutal" were given at 11.30 p.m., as she was restless.

The First Labour.

The membranes ruptured spontaneously at 1 a.m. on February 4, and within an hour contractions were occurring. It had been noted on January 1 that one fœtus was now presenting by the vertex, whilst two continued to present by the breech. The presenting part was the head of the vertex, fully engaged in the pelvis. On January 31 a second fœtus was found to have turned also, its head lying to the right above the pubis. The third was still presenting by the breech. Thus they remained during this "first labour", which was entirely normal. At 4.15 a.m. she was given four drachms of "Mother's Mixture" (Mayes, 1950), and at 5.30 a.m. an intramuscular injection of 100 milligrammes of pethidine.

At 6.45 a.m. the head was crowned (occipito-anterior position) and was delivered normally, without forceps, under light "open" chloroform anæsthesia. The perineum and vagina were not lacerated. The child was a vigorous female, weighing six pounds eight and a half ounces.

The maternal end of the umbilical cord was identified as belonging to the first-born, then wrapped in a sterile pad to protect it from contamination. Maternal blood loss was negligible, the blood pressure was 145 millimetres of mercury, systolic, and 100 millimetres, diastolic, and a catheter specimen of urine contained "one-twelfth" of albumin.

The Interval.

There followed an interval of nearly twelve and a half hours before the proper resumption of labour. To prevent anxiety during this period, the mother was informed that there might be "two labours with an interval between". She readily accepted this idea. She was well and showed no distress or anxiety, but was tired and wanted to sleep.

Abdominal palpation showed that the head of the second fœtus was now engaging in the pelvis. The third was still presenting by the breech, its head being easily palpated below the mother's right costal margin.

A few weak, irregular uterine contractions occurred, with backache, but the presenting part on rectal examination remained high. At twelve noon there was a sudden gush of liquor, necessitating a change of bed linen. Nothing else happened; the cord did not prolapse, neither did the head descend.

At 1 p.m. the urine contained "one-third" of albumin, and the blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic; the patient's face looked puffy, and she had no desire for food or drink, despite the fact that she had had none since labour began. An intravenous drip administration of dextrose solution, 25% in water ("Soluvac") was started at 1.30 p.m. and discontinued at 2.20 p.m.; during this period the patient received 600 cubic centimetres of solution. After this, backache disappeared altogether and she slept soundly for three hours. She woke at 5.30 p.m., and during the next half-hour drank several glasses of "Akta-Vite" in milk. The urine now contained "one-sixteenth" of albumin. She appeared refreshed and cheerful, and was still unworried by the temporary cessation of activity. Indeed, her confidence and good spirits were infectious.

The Second Labour.

Contractions returned spontaneously that evening at 7.10 p.m. By 9 p.m. she was in strong labour and beginning to bear down. At this stage she showed distress for the first time. On rectal examination the head was found to be still above the ischial spines, the os was half dilated, and (despite the copious gush at mid-day) the forewaters were intact. They were artificially ruptured at 9.20 p.m. The patient continued to push strongly, but thereafter was not distressed. An intramuscular injection of 50 milligrammes of pethidine was given at 9.55 p.m. and again at 11.15 p.m. The second baby (occipito-anterior position) arrived rapidly and without assistance at 11.40 p.m. and cried lustily. It was a female, weighing six pounds twelve ounces. The maternal end of the cord was identified as belonging to the second baby.

There was no repetition of the earlier delay. The remaining fœtus, hitherto presenting by the breech, had undergone spontaneous version as the second was born, and its head was found to be well down in the pelvis. At 12.5 a.m. there was a strong contraction and the membranes bulged externally. They were ruptured with the

next contraction at 12.10 a.m., and the head (occipito-anterior position) was born immediately after. The mother then received an injection of one cubic centimetre of ergometrine intravenously, and delivery of the baby was completed. This also was a female and weighed five pounds thirteen and a half ounces. She cried vigorously at birth. The maternal end of the cord was identified as that of the third baby.

The Third Stage.

The duration of the third stage was five minutes, and there was practically no bleeding. The placenta, weighing three pounds four ounces, was easily expressed after obvious signs of separation. The mother was very well indeed. However, since her hæmoglobin value before delivery was only 9·7 grammes *per centum*, a transfusion was considered desirable and one pint of fresh blood was given by slow drip administration.

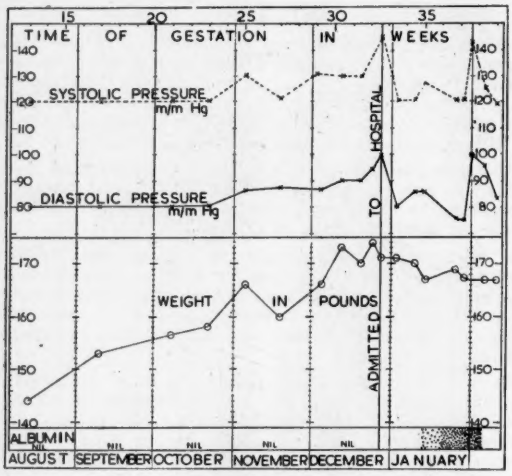

FIGURE II.

The Puerperium.

The puerperium was uneventful, and the babies were strong and caused no anxiety. They were all discharged from hospital on February 13, at the mother's insistence. This capable young woman has managed them without fuss and without help, and has required no professional advice on how to feed them. Their progress has been completely normal. The first and third, in order of arrival, are identical brunettes, the second is a blue-eyed blonde. These appearances are consistent with Dr. George Sippe's report on the placenta, which appears below.

The Placenta.

The placenta with three cords attached was submitted for examination to Dr. George Sippe, of Maitland. On the maternal aspect the specimen is seen to consist of two separate placentæ. One measures 17 centimetres in diameter, and it is from this that the cord marked "two" arises. The cavity of this placenta is separated from its companion by four distinct membranes, which would indicate a separate origin for the second child. The remaining placenta measures approximately 21 centimetres across, and from it two cords arise—"one" and "three". The placental sacs here appear to be separated by two layers only, which indicates uniovular origin. No macroscopic abnormality was noted in either placenta. This arrangement of one double placenta with one chorion and two amnions, and a completely separate single placenta is by far the commonest arrangement in triplet births (Novak, 1947).

Comment.

Should one interfere by rupturing the membranes after the first delivery or wait until Nature resumes the offensive? In a case such as this the period of waiting can be an anxious one. On the other hand, is there any reasonable certainty that interference will be as effective for the second triplet as it is for the second twin? There seems to be no very definite opinion about this. If there is, it does not appear in any of our text-books, in which discussion is limited to the management of twins. This is understandable, as the triplet problem is rarely encountered, and even then delivery is usually rapid and requires no plan of attack. But it can be disconcertingly slow, as with quadruplets also (Elliott *et alii*, 1951).

The feature of great interest in this case is the long interval of rest between two periods of labour—labour so strong that the term "uterine inertia" cannot be applied to any part of this confinement, if by inertia we mean inefficiency or exhaustion. Here was efficient inactivity for a time: Nature's way, no doubt, of preventing exhaustion.

Provided sepsis can be prevented, the courage of the obstetrician sustained, and early vaginal examinations avoided, non-interference has much to recommend it.

Acknowledgements.

We are indebted to Dr. T. W. Anderson, of Newcastle, for the skiagram, to Mr. V. H. Geary for the excellent photographic reproduction, and to Mr. L. W. Gill for preparing the chart of weight and blood pressure.

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WHITE SPONGE NÆVUS OF THE MUCOSA (CANNON), NÆVUS SPONGIOSUS ALBUS MUCOSÆ: REPORT OF A CASE.

By L. G. ABBOTT AND L. A. MUSSO,
Sydney.

Clinical Record.

MR. A, aged seventy years, of Scandinavian descent, in July, 1954, complained of the recurrence inside his left cheek of a lump which had been excised eleven years previously and examined histologically (1943). Shortly after its removal he felt that it was growing again, and it steadily increased in size to assume its original proportions. At no stage had it been painful, nor did it cause any irritation other than by its bulk. The patient stated that he drank alcohol rarely, but had smoked a pipe since adolescence. He could give no relevant history of his parents, but upon request communicated with his children and immediate family, and no skin rashes or tumours were disclosed.

On examination of the patient, the lesions were restricted to the oral mucosa, the rectal and laryngeal mucosæ being clear. The Wassermann and Kahn tests produced negative results. The buccal mucous membrane appeared paler than is usual and in some places presented a milky sheen. On the upper surface of the tongue, in the mid-line, were numerous brown, hairy patches.

The lesion appeared on the buccal surfaces of the cheeks as a band about 1·5 centimetres wide, extending anteriorly from opposite the third molar at the level of closure of the teeth; at the angles of the mouth each band forked and ran onto the lateral third of the buccal surface of the lips. Narrower, but similarly formed, bands were

visible on the anterior third of the under-surface of the sides of the tongue. The colour was decidedly milky-white, the depth of colour varying only with the thickness of the lesion.

On the left cheek, the band at the back was quite flaccid and undulant, apparently owing to the laxity of the tissues. As it progressed forwards, it became thickened and formed raised small plaques, in which numerous orifices, divided by extremely fine membranous lines, were visible. Just behind the angle of the mouth a single large plaque was apparent, of rhomboidal shape, sharply defined, with steep edges, the surface of which was raised about 0·5 centimetre above the surrounding mucosa. The plateau-like summit was broken up by numerous depressed lines into small rhomboidal areas, which were pitted, giving a honeycomb appearance; on palpation it yielded a sensation of rubbery firmness. The resemblance of this plaque to a white sponge was unmistakable, and no more apt name could be applied to it (see Figure I). In its other situations this pattern was reproduced, but the formation was not so far advanced. A biopsy of the lesion on the left buccal mucosa was carried out in July, 1954.

FIGURE I.
Part of the lesion in 1954, on the buccal mucosa of the left cheek, showing its sponge-like surface.

The histological appearances are the same in the lesion examined in 1943 and in the 1954 biopsy; but as that of 1943 shows them more distinctly, it is described as follows. There is a pronounced acanthosis in the form of several "peg"-like processes pushing down in the corium (see Figure II). These pegs are separated from one another, usually by the *stratum Malpighii* in the deeper parts and by thin lamellæ of parakeratosis in the superficial parts. The external surfaces of these pegs are in the shape of cup-like depressions whose bases are composed of pale-staining, very large squamous cells of varying size, the nuclei of which are surrounded by a clear halo (see Figure III). The majority contain granules of varying size. The granular layer is absent, since it is a mucous surface. The deep parts of these pegs consist of hypertrophied squamous cells, and there may be evidence of vacuolation, pale granular cytoplasm or both.

No significant changes are detectable in the corium.

Summary.

This rare condition has been reported in the literature only twice previously, the first record being that of Cannon, under the title we have applied to our case. Ludy and Shirazy described the condition as *leucokeratosis oris*. In each of these reports a definite family history was present, and Cannon was able to show that tobacco smoking appeared to play no part in the formation of the lesions.

The singular appearance of the lesions and their localization to mucous membranes seem to distinguish the condition from the ichthyosiform group of nævi.

Acknowledgement.

We should like to thank the photographic department, Sydney Hospital, for the photographs and photomicrographs.

References.

CANNON, A. B. (1935), "White Sponge Nævus of Mucosa (Nævus Spongiosus Albus Mucosæ)", *Arch. Dermat. & Syph.*, 31: 365.

LUDY, J. B., and SHIRAZY, E. (1941), "Congenital Leukokeratosis Mucosa Oris (Keratosis Disséminée de Muqueuse)", *Internat. Clin.*, 4: 38.

Legends to Illustrations.

FIGURE II.—Part of the lesion examined in 1943; hæmatoxylin and eosin stain, × 22. The peg-like processes of epidermis pushing down into the corium, together with their cupped external surfaces, are well seen.

FIGURE III.—Higher power of the inset in Figure II (× 120). Note the base of a cup-like area, in which the pale staining squamous cells are visible.

Reviews.

Genital Prolapse and Allied Conditions, by Percy Malpas, Ch.M. (Liverpool), F.R.C.S. (Eng.), F.R.C.O.G.; 1955. London: Harvey and Blythe, Limited. 9″ × 6″, pp. 200, with 37 illustrations. Price: 47s. 6d.

THE author is a well-known authority on all aspects of genital prolapse. It was he who suggested the Liverpool work done on the vesico-urethral junction in stress incontinence.

The contents of this book offer a very fair summary of the whole condition, for the cure of which the gynæcologist probably gets more thanks than any other therapeutic procedure. Malpas is well qualified to speak because this work is based on experience gained from over 7000 operations. Over and over again the anatomy and physiology of the region are referred to, and the physiological principle of repair is adhered to where possible.

An unusual and highly commendable feature of the book is the *verbatim* quotations from many British gynæcologists, notably Donald, Fothergill, Shaw and Bonney.

If any criticism is to be offered, it is perhaps that there are not enough illustrations in the text—for instance, not one appears in the section dealing with vaginal hysterectomy. Nor is there any discussion on the types of anæsthesia used during these repairs, a notable omission perhaps because many surgeons are convinced of the value of low spinal anæsthesia.

The ætiology of genital prolapse is discussed from a commonsense viewpoint, and it is pointed out that episiotomy is not the certain prophylactic measure that current fashion would have us believe. Nor is the long trial labour, conducted too conservatively, always the obstetric triumph it might appear to be. As distinct from these acquired causes, tissue weakness is discussed in some detail.

In the discussion of the varieties of genital prolapse a working classification is submitted by the author, in an endeavour to clear up the confused nomenclature. But all gynæcologists will agree with him that each patient must be treated as an individual and distinct problem, and the curative operation varied accordingly. Coming from the pen of this author, it is only natural that the Fothergill or Manchester operation be given first place. Moreover, this corresponds with the general usage of gynæcologists in this country, and it will be of interest to them to read quotations from the original papers to help determine who actually first promulgated the principles underlying this operation. The description of the technique of the operation is full and easy to follow. It is good to see stress laid on the importance of ensuring an adequate length for the anterior vaginal wall, because it is the failure of the operator to do this which brings this operation into some disrepute, especially by those who advocate vaginal hysterectomy in almost every case.

Should the gynæcologist be unfortunate enough to damage ureter, bladder, urethra or rectum, a full description of each necessary repair is given.

This excellent book is completed with a section dealing with the complications of repair operations, including post-operative bleeding and infection, and a discussion of the special problems of the disturbances of micturition, the

rectal complications of prolapse and a brief summary of retroversion of the uterus in relation to this.

While this book will be of value to the occasional gynæcologist, it is recommended as an essential in the library of the specialist.

Hypnotic Suggestion: Its Role in Psychoneurotic and Psychosomatic Disorders. A thesis by S. J. Van Pelt, M.B., B.S.; 1955. Bristol: John Wright and Sons, Limited. 7¼" x 5", p. 96. Price: 8s. 6d.

THIS little book is written in the same style as the author's other popular books on hypnosis. In it he claims originality for a new theory as to the ætiology of the psychoneuroses; a new method of treatment for the psychoneuroses; and a new theory as to the nature of the hypnotic state. The author is to be congratulated on his courage, and it is gratifying to read of anyone attempting original work, especially in the direction of finding shorter methods of treatment for nervous illness.

The author groups all the psychoneuroses together, and postulates a common cause. At some time in the past the patient has spontaneously gone into a state of autohypnosis following some emotional incident, and events at the time have become impressed on the mind in the same way as a post-hypnotic suggestion. Treatment involves three principles: relaxation, realization and reeducation. Relaxation is obtained in light hypnosis, and the post-hypnotic suggestion is given that the cause of the trouble will come to the patient's mind before the next session. The patient reports back with some significant thought, and reeducation proceeds with sessions of hypnosis and the suggestion of healthier attitudes.

The author claims an original theory for the nature of hypnosis as a form of super-concentration of the mind.

The approach is simple and direct. It is a readable little book for those who have no wish to be troubled with the complexities of psychodynamics.

Notes on Books, Current Journals and New Appliances.

The London Medical Handbook; 1955. London: The British and Colonial Druggist, Limited. 10" x 9", pp. 126, with five illustrations. Price: 10s. 6d.

MOST of this handbook is taken up with a list of drugs, listed by their proprietary names, each with the maker's or supplier's name, the composition of the drug and the therapeutic indications for its use (presumably as defined by its manufacturer or supplier). A "therapeutic index" provides a useful form of cross-reference. Short contributed articles deal (rather too briefly) with recent advances in the fields of therapeutics, surgery, obstetrics and gynæcology, and *materia medica*.

The Work of the Mental Nurse; a survey organized by a Joint Committee of the Manchester Regional Hospital Board and the University of Manchester; 1955. Manchester: University Press. 8½" x 5½", pp. 164, with many illustrations. Price: 10s.

THIS report of a Joint Committee on mental nursing is an analysis of conditions in mental hospitals in Great Britain, and of the scope and allocation of mental nursing duties. It is an excellent treatise on organization and management, and contains some valuable recommendations on how to make the best use of nursing staff to secure efficient results.

Collected Papers on Aviation Medicine, presented at Aeromedical Panel Meetings of the Advisory Group for Aeronautical Research and Development, Palais de Chaillot, Paris; 1955. London: Butterworth's Scientific Publications. 10" x 6", pp. 218. Price: 37s. 6d.

AT the Aeromedical Panel Meetings and at General Assemblies of the Advisory Group for Aeronautical Research and Development, various papers are delivered on subjects of particular and general interest. The 18 papers collected in this volume cover a wide field. The correlation between medical research and aeronautical technique, discussions on the effect of cold, of noise, and of abrupt deceleration on the human body and therefore the modifications which these must impose on aircraft design, tests for discovering latent epilepsy in potential pilots, problems of survival in the Arctic, are all matters of direct interest to aviation. Also, many of them are of general medical interest in some way. However, this volume is chiefly of value to those connected with aviation.

Synopsis of Audiology, edited by William M. Fitzhugh, jnr., M.D., C.M., D.Sc. (Med.); 1955. California: J. W. Stacey, Incorporated. 11" x 8½", pp. 88. Price:

PRINTED in manuscript style, this synopsis consists of a series of questions and answers, from Number 1, which defines audiology, to Number 345, which describes the physiology, cause and treatment of a persistent falsetto voice. It is intended as a guide for those being introduced to the science of hearing, and within its very wide limits it covers a very large field.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"How to Look at Illness", by Norbert Glas, M.D. (Vienna), L.R.C.P. (London), M.R.C.S. (England). London: New Knowledge Books. 7" x 4½", pp. 48. Price: 3s.

Based on the philosophical work of Rudolph Steiner.

"The Human Soul: In Sleeping, Dreaming and Waking", by F. W. Zeylams Van Emmichoven, M.D. London: New Knowledge Books. 7" x 4½", pp. 24. Price: 2s.

Based on the philosophical work of Rudolph Steiner.

"Textbook of Clinical Pathology", edited by Seward E. Miller, M.D.; Fifth Edition; 1955. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 6", pp. 1237, with illustrations. Price: £6 1s.

Completely revised with a number of new chapters.

"Modern Operative Surgery", edited by the late G. Grey Turner, LL.D., D.Ch., M.S., F.R.C.S., F.R.A.C.S., F.A.C.S., and Lambert Charles Rogers, V.R.D., M.Sc., M.D., F.R.C.S., F.R.C.S.E., F.R.A.C.S., F.A.C.S., with a foreword by Sir Gordon Gordon-Taylor, K.B.E., C.B., M.A., LL.D., Sc.D., M.D., M.S., F.R.C.S., F.R.C.S.E., F.R.C.S.I., F.R.A.C.S., F.A.C.S.; Fourth Edition; 1955. London and Sydney: Cassell and Company, Limited. Volume I. 9" x 6", pp. 1249, with many illustrations. Price: £5 5s.

This edition was commenced by Professor Grey Turner and completed by Professor Lambert Rogers after the death of the former.

"Clinical Psychiatry", by W. Mayer-Gross, M.D. (Heidelberg), F.R.C.P. (Lond.), Eliot Slater, M.A., M.D. (Camb.), F.R.C.P. (Lond.), D.P.M., and Martin Roth, M.D. (Lond.), M.R.C.P. (Lond.), D.P.M.; 1955. London and Sydney: Cassell and Company, Limited. 9½" x 7½" pp. 670, with 16 plates. Price: £3 15s.

A text-book which aims to include recent developments and to be of use to the student, the general practitioner, the non-psychiatric medical consultant and the psychiatric consultant.

"Modern Treatment Year Book 1956: A Year Book of Diagnosis and Treatment for the General Practitioner", edited by Sir Cecil Wakeley, Bt., K.B.E., C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S.; 1956. London: Published for the Medical Press by Ballière, Tindall and Cox, Limited. 8½" x 5½", pp. 352, with illustrations. Price: 25s.

Contains 37 articles by various authors.

"Medical Terms: Their Origin and Construction", by Ffrangcon Roberts, M.A., M.D., F.F.R.; Second Edition; 1956; London: William Heinemann (Medical Books), Limited. 7½" x 5", pp. 96. Price: 6s.

The title indicates the contents of this book, which is not a dictionary.

"Ten Patients and an Almoner", by Flora Beck; 1956. London: George Allen and Unwin, Limited. 7½" x 5", pp. 159. Price: 12s. 6d.

The author is a qualified almoner who has held various hospital and university appointments and has had over fifteen years' practical experience of medical social work.

The Medical Journal of Australia

SATURDAY, APRIL 28, 1956.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words of phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

REPORT ON THE HEALTH OF GREAT BRITAIN.

In the issue of January 14, 1956, Part I of the report of the Ministry of Health of Great Britain for the year ended December 31, 1954, was discussed in these columns. Part I was presented by the Minister of Health to Parliament in September, 1955, and Part II, which has recently come to hand, was presented to Parliament in December, 1955. It will be remembered that Part I dealt with the National Health Service and with welfare, food and drugs and civil defence. Part II deals with the state of the public health, and is the annual report of the Chief Medical Officer for the year 1954. In the beginning of his covering letter to the Minister of Health, the Chief Medical Officer, Sir John A. Charles, states that in 1954 more settled conditions of life were continued with the ending of the rationing of fat and meat. Full employment was obtained, wages again rose and there was general industrial prosperity. The vital statistics for the year show little change on those of previous years, though the steady fall in infant mortality rate has continued. The rate for 1954 was 25·4 per 1000 related live births; the neonatal mortality rate was 17·7 and the post-neonatal mortality rate was 7·7. In 1953 the infant mortality rate was 26·8. The infant mortality rate of England and Wales and of different countries is set out in a table. The rate for England and Wales has fallen steadily since 1921-1925, when it was 76. For 1954 the lowest infant mortality rate was that of Sweden, 19; the position of Australia is the same as that of New Zealand, 22. The Netherlands and Canada have a figure of 21. The highest figures are Italy, 53, France, 41, and the Irish Republic, 38. There was an increase in population of 165,000 from mid-1953; at mid-1954 the total was 44,274,000. With the increase in population there has been an increase in the proportion of those aged sixty-five years and more. In the higher age groups women are substantially more numerous than men. It is pointed out that these two features, which are likely to persist for a considerable time, hold implications for the future of the health and welfare services.

Stillbirths, we read, continue to present an obstinate problem. They have shown none of the steady decline manifest in infant mortality generally, and "it is clear that until more is known of their causation little improvement can be expected". From the period 1936 to 1940 to the period 1946 to 1950 the mean annual stillbirth rate fell from 38 to 24 per 1000 total births. This is thought to have been due to the cumulative effect of improved ante-natal care, and possibly to that attention to the nutrition of expectant mothers which became an article of national policy during the war and afterwards. These causes seem no longer to affect the trend to the same extent, and it is thought that new measures will have to be brought to bear. Unfortunately no analysis has been made of maternal deaths for 1954. At the time when this report was written reports had been received on nearly 1400 deaths due to or associated with pregnancy and childbirth and occurring in the period 1952 to 1954. This group of deaths is being studied as a whole. It is known that there is a wide variation in the extent and quality of ante-natal care, especially in the measures taken for the detection of the early signs of toxæmia of pregnancy. Some 9% of obstetric deaths are due to eclampsia, and we are told that when to these are added the 13% attributed to the ante-partum hæmorrhage which is causally associated with it "we begin to appreciate how important these conditions are and how valuable any improvement in their prevention would be". It would seem justifiable to assume that there is room for one or more of the following: (*a*) post-graduate education of general practitioners in ante-natal care and the prophylactic treatment of eclampsia; (*b*) the education of midwives, who undertake the greater part of the midwifery in England and Wales; (*c*) the education of patients in the need for ante-natal care and proper obstetrical supervision.

In regard to causes of death the group of cardio-vascular and renal diseases (diseases of the heart and circulation, vascular diseases of the nervous system and nephritis) accounted for 263,425 or 52% of the total 501,896 deaths in England and Wales in 1954. Cancer in its various forms was responsible for 18% of the deaths. Influenza, pneumonia, bronchitis and other respiratory diseases, excluding tuberculosis, caused 10% of the deaths. The three groups of diseases mentioned contributed 81% of the total mortality. In order of numerical importance other causes and groups of causes individually responsible for more than 0·5% of all deaths at all ages were as follows: accidents other than those associated with motor vehicles, 2·2%; tuberculosis in all its forms, 1·6%; ulcer of the stomach and duodenum, 1·1%; suicide, 1·0%; motor vehicle accidents, 0·9%; congenital malformations, 0·9%; hyperplasia of the prostate, 0·9%; all diseases classified as infective other than tuberculosis, gastro-enteritis and diarrhœa, 0·8%; *diabetes mellitus*, 0·6%. It is notable that of the 90,095 deaths from all forms of cancer in 1954 the proportion attributed to cancer of the lung was 18%; in

1953 and 1952 the figures were 17% and 16% respectively. The total number of deaths from cancer of the lung in 1954 was 16,331, or 1199 more than the total in 1953. There is stated to be evidence that though the mortality among males above the age of fifty-five years continues to increase, the rates at ages below this have become relatively stable, and that further increases in the number of deaths will be found only among the older age group. The comparative mortality indices from 1931 to 1933 for all sites of cancer indicate that mortality from cancer as a whole has been slowly declining among women, but steadily increasing among men. Male increase is accounted for by the rise in the death rate from cancer of the lung. In regard to major cardio-vascular and renal diseases (including nephritis in all its forms), there were more deaths from vascular lesions affecting the central nervous system than there were from arteriosclerotic heart disease including coronary disease. Hypertension with heart disease caused a little more than one-fifth of those caused by arteriosclerotic heart disease including coronary disease. A group called "other heart disease" headed the list of deaths from cardio-vascular and renal diseases. In the group of respiratory diseases there were relatively few deaths from influenza. The absence of an influenza epidemic also led to fewer deaths from pneumonia and bronchitis. It is pointed out that more deaths occurred in 1954 than in 1953 from accidents, from duodenal and gastric ulcer and from suicide, which is regarded as the most useful single index to mental health provided by mortality statistics. These three causes are becoming important fields of inquiry in preventive medicine, but their mortality has not substantially changed in recent years. It is unsatisfactory, however, that the total deaths from motor vehicle accidents, from other accidents and from suicide have shown increases for two years in succession.

In the reference to diphtheria some important observations are made. The disease has ceased to be a public health problem in the sense of being dangerous to large numbers of persons every year, but its control remains an important function of public health authorities. An appreciable slackening of the immunization campaign might be followed by serious consequences. Immunization of children not only prevents large numbers from contracting diphtheria; it also mitigates the severity of the infection if the child should fall a victim. It was found among the notified cases in 1954 that there were no deaths among 40 children who had been immunized before their illnesses occurred, whereas among 60 children who had never been immunized there were six deaths. These facts are, of course, well known, but they belong to those which a general practitioner may use in the preventive medicine instruction which he gives to his patients. Stress is laid on the importance of the booster dose which it is recommended should be given five years after actual immunization has been carried out. Reference is made to the cardiac and other delayed effects of diphtheria, which can increase to a considerable extent the fatality ratio of adults which is calculated on the basis of annual notifications. "The certification procedure by which a death from delayed effects many years after is attributable to the original acute disease is a cause of difficulty in interpreting the mortality figures of other infectious diseases besides diphtheria, but it is nevertheless in accordance with current international procedure which is, however, now under review."

The number of notifications of scarlet fever during 1954 was the lowest yet recorded. The medical profession is warned that although scarlet fever seems to have lost its former capacity for causing death, what happened in earlier years should be remembered. At Tours in France scarlet fever was a very mild disease from 1799 to 1822, and no death was recorded from it. In 1824, however, an epidemic broke out which proved so deadly and refractory to treatment that one observer regarded the disease as being as formidable as plague, typhus or cholera. There is no guarantee that the severe form of the disease will not return. It is not unwise to suggest that watchfulness is indicated.

There was a substantial decrease in the number of corrected notifications of measles during the year, the total 147,000 being the lowest since notification was introduced fourteen years ago. In measles complications and deaths are largely caused by secondary bacterial invasion, and conditions affecting the respiratory system, particularly bronchopneumonia, account for more than three-quarters of the deaths. Mention is made of some work by G. I. Watson, who used sulphadiazine with success in the prophylaxis of complications. We are told that in Watson's experience chemotherapy was of value for children showing signs of preexisting bacterial infection when the sulphonamide acted curatively. The incidence of complications, however, was greater when sulphonamide was given to the children who were apparently free of signs of bacterial infection at the onset of their illness. This experience is compared with that of H. S. Banks, who found that the routine administration of sulphonamide for six days from the appearance of the rash greatly reduced the incidence of complications; when the sulphonamide treatment was combined with appropriate treatment of any complications present when the child was admitted to hospital it was associated with a fall in the case fatality rate to less than one-tenth of what it had been in previous epidemics. It is noteworthy that the threatened attack of measles can, when it is judged advisable, often be averted or modified in known contacts by the appropriate use of γ globulin. The College of General Practitioners has instituted a two-year study of the value of sulphonamides and antibiotics in the prophylaxis of bacterial complications of measles. The result of this investigation will be awaited with interest.

The position in regard to poliomyelitis was satisfactory in 1954, the number of notifications being the smallest since 1948. It is stated with justification that poliomyelitis is a disease whose capacity to arouse dread bears little relation to its frequency and fatality. At the same time, although the authorities in England and Wales see poliomyelitis in its proper perspective, they remark that it cannot be regarded lightly. There always comes to mind the grim experience of Denmark, where out of a clear sky an epidemic descended in 1952 causing 5676 cases, of which 2450 were paralytic and 262 fatal. The research being undertaken by virologists in regard to a vaccine is mentioned, and we know, of course, what has been recently achieved in that field.

In regard to acute infective encephalitis it is pointed out that the disease is difficult to diagnose during the life of

a patient, and after death it can be identified only by a careful examination of the nervous tissues by a pathologist skilled in the interpretation of abnormal appearances of the brain and spinal cord. The question is asked why it should continue to be notifiable. The answer is given that it is better to know some of the cases which occur than not to know at all. Notification always keeps doctors and nurses aware of the disease, and in this way provides for the recognition of cases which might otherwise be missed.

There has been a large increase in the number of cases of dysentery notified. The number of cases notified was over one and a half times that notified in 1953 and was the highest on record. Fortunately the severity of the disease was not great. The epidemiological evidence suggested that it was largely transmitted directly from person to person and not by contaminated food. In connexion with this disease a sermon on personal cleanliness might be preached.

There are several other aspects of this report which should receive passing mention. In the account of the work of the National Blood Transfusion Service a reference is made to what appears to be extravagant use of blood in modern hospital practice. The Old Country is not alone in having to consider this matter. It is pointed out that in 1954 the regional transfusion centres issued more blood than in any previous year, and the total was slightly more than three times that used in 1946. Altogether hospitals received nearly 620,000 donations of blood collected by the National Blood Transfusion Service. The question is asked whether the steady increase in the use of blood from year to year is entirely justified, since the number of hospital beds is not increasing and the average stay of the patient in hospital is not diminished at rates which would explain more than a small part of the increased use of blood. It is pointed out that the value of plasma is insufficiently appreciated.

Another point which seems to be shared by other countries is that the recruitment of suitable youths for training as laboratory technicians continues to exercise the mind of pathologists. It will be interesting to see whether the subcommittee appointed by the Central Pathology Committee to study the question will come to any useful conclusions. Sections of the report which cannot be dealt with at present include nutrition, the adrenal corticosteroids, rehabilitation, the care of old people and nursing and the public health. Possibly on some future occasion these subjects may be referred to in these columns.

Current Comment.

THE EVALUATION OF HYPOTENSIVE DRUGS.

In any disease in which non-pharmacological factors strongly affect the condition of the patient, and his symptoms and physical signs, the evaluation of any treatment with drugs is very difficult. Hypertensive vascular disease is such a condition, and many pitfalls are created by variables, other than drugs, that are always present when one attempts to determine the effect of any drug. These factors are commonly neglected; hence largely the differences of opinion expressed by different observers on the effect of any particular drug on the disease condition. New drugs have become available during the past few years which, it is claimed by some, are valuable in the treatment of hypertension. Other observers have found one or several of these drugs to have little or no value or to have had side effects which more than counterbalance any good effects.

A. P. Shapiro,[1] in a very extensive study, has endeavoured to measure the effects of the non-pharmacological factors in relation to the effects of the drugs. The hypotensive agents studied were hydralazine ("Apresoline"), hexamethonium, rauwolfia, veratrum alkaloids, and low sodium diets. A large number of patients, some in hospital, some out-patients, with hypertensive disease in all stages of severity, were examined. The conditions under which the patients were examined were standardized as closely as possible to cut down the number of variables, and every effort was made to ensure that the assignment of drugs and placebos was made as random as possible and to keep from the ward physicians, nurses and patients the nature of the drug being used.

The factor with the biggest influence is hospitalization. Blood pressure levels usually decline after the patient's admission to hospital, but the effect is very variable in different patients. Of 33 patients who were put into hospital for study the lowest blood pressure was attained after a minimum of three and a maximum of twenty-eight days, no specific drugs being given. The magnitude of the fall was from six to 85 millimetres of mercury mean blood pressure. Another factor of importance is the doctor-patient relationship—for example, the effect on the patient of changes in the enthusiasm of the doctor for a particular drug. Life changes are also important but difficult to assess.

The symptoms of hypertensive vascular disease are largely non-specific, and the relief of headaches, malaise and easy fatigability which many patients exhibit under treatment is unreliable as an index of improvement. Most patients show an overall improvement in well-being when any new treatment is instituted.

It is not possible here to discuss the methods which were used to evaluate and allow for the effects of the non-pharmacological factors, but they seem to have been very effective in enabling an evaluation of the various hypotensive drugs to be made. The usual method of determining the clinical efficiency of a given drug consists of comparison of its effects in closely identical groups of treated and untreated patients. Hypertension shows so many different types and manifestations, with ill-defined criteria of severity, that the selection of comparable groups is practically, if not entirely, impossible. Shapiro has used each patient as his own control, and the blood pressure during a period of drug administration is compared with that during a period with no medication.

As a result of this survey Shapiro has arrived at certain opinions as to the value and proper place in treatment of the hypotensive drugs commonly available. He considers that these agents can lower blood pressure and, in individuals with severe, rapidly progressive forms of the disease, in whom papilloedema and encephalopathy are present, and yet renal function remains in good condition, an intensive attempt to lower the blood pressure is warranted. One must, however, be on the watch for detrimental effects of the drugs on blood flow to vital organs. At best the treatment slows rather than halts the disease process. Where there is severe renal damage it is doubtful if any of these drugs can improve conditions, and they can certainly worsen them. In the individual with hypertension with little or no evidence of progression of the process it is doubtful whether these drugs have any significant value. "Development of rational therapeutics still awaits the acquisition of additional knowledge of etiology and pathological physiology. Until such time we can at least avoid obscuring the problems further by credulous interpretations of therapeutic results."

At a clinical conference held by H. A. Schroeder and H. M. Perry and reproduced in detail in the January, 1956, issue of *Circulation* a review is given of the usefulness of hypertensive drugs in the treatment of hypertension based

[1] *J.A.M.A.*, January 7, 1956.

on extensive clinical use of the drugs. As the discussion is very detailed, a summary will be given of the more important points. The different drugs act on different parts of the body, and all produce unpleasant side effects. Thus hexamethonium and similar drugs are ganglion-blocking agents, reserpine is a cortico-hypothalamic depressant, veratrum alkaloids influence vasomotor centres in the medulla, and hydralazine acts on constricted vascular smooth muscle. For the milder cases one of the drugs alone, such as reserpine, may produce good results; in more severe cases two or three different drugs having different actions are needed. All the drugs must be used with great care and frequent observation. The patients should be taught to take their own blood pressures. Initially drug-induced hypotension is not rare and is alarming but apparently not dangerous. It may take weeks or months before the inconveniences resulting from the drug treatment pass off or become tolerable. The greatest dangers to life have involved cessation of therapy in severe and malignant cases. In many cases of all degrees of severity the sudden lessening of the dose or cessation leads to a high blood pressure which requires much larger doses of the drugs to bring again to near normal. Beneficial effects of lowering blood pressure can be definitely established only for the small group of patients in the "malignant" phase of hypertension, as characterized by a mean diastolic pressure in excess of 130 millimetres of mercury, severe renal dysfunction and retinitis with œdema of the optic disks. Adequate treatment with the new drugs may stave off the end for many months with reasonable comfort to the patient. In cases of symptomless hypertension the doctor and patient have to decide whether the favourable results to be expected sufficiently counterbalance the unpleasant side effects of the drugs. In the more severe cases the same decision has to be made with a bias in favour of treatment. A non-protein nitrogen level in the blood much greater than 100 milligrammes per 100 millilitres contraindicates the use of any of the drugs, for diminution of glomerular filtration pressure often quickly proves fatal. The different drugs should never be given in combination, for the dose of one may be constant and that of another variable. Schroeder, in his concluding remarks, writes: "Modern antihypertensive drugs require intelligent handling by both physician and patient. When so employed the results are usually gratifying to the patient. As a first approximation of specific treatment, they are the best we have. . . . There are enough drugs now available to control the hypertension and prolong longevity of almost any patient who wants to be treated and will submit to therapeutic inconveniences." One of the physicians at the conference quoted Sir Robert Hutchinson as praying that we might be delivered "from inability to let well enough alone, from too much zeal for the new and contempt for the old, . . . and from making the cure more grievous than the ill".

LEUCHÆMIA RESEARCH.

Early in 1953, Sir Henry Dale, Professor L. J. Witts and Dr. F. H. K. Green suggested that it would be valuable to hold an informal discussion at an international level on the present position of research in leuchæmia and the directions in which further research might most profitably be pursued. They put forward this proposal on behalf of the (European) Scientific Advisory Committee of the Lady Tata Memorial Trust, a charitable Trust founded by the late Sir Dorabi Jamseti Tata, of Bombay, in memory of his wife. This Trust has become world-famous in the last twenty-one years for its support of research on diseases of the blood and especially leuchæmia. The Director of the Ciba Foundation agreed to organize such a symposium, and the actual meeting was held in November, 1953. The report of this meeting, now published in book form,[1] consists of a series of papers read at the meeting, with reports of discussions on each paper. A list of the thirty-five members participating in or attending the symposium includes such well-known names as M. Bessis, of the National Blood Transfusion Centre, Paris; J. Craigie, of the Imperial Cancer Research Fund, London; J. Englebreth-Holm, Copenhagen; Astrid Fagraeus, Stockholm; A. R. Gopal-Ayenfar, Bombay; M. C. G. Israels, Manchester; L. O. Jacobson, Chicago; S. Moeschlin, Zurich; Bo Thorell, Stockholm; and Sir Lionel Whitby, Cambridge.

In his opening remarks the chairman, Dr. L. J. Witts, of Oxford, said that while the study of diseases of the blood was of intense interest in its own right, it was impossible for the medical researcher to be unaware of the human and emotional problems involved. Few diseases were more tragic than leuchæmia or more inexplicable in terms of human suffering. A generation ago it might have been regarded as a relatively rare and unimportant disease, but this was no longer the case, for leuchæmia now accounted for more deaths in childhood than all the common acute infections put together, and it formed a significant portion of malignant disease in later life. In animal husbandry likewise it took an important toll of flocks and herds. Steady progress had been made in the understanding of leuchæmia, and there had been some small but measurable improvement in methods of treatment. The difficulties of research in leuchæmia had not prevented some first-class workers from entering this field. It was felt, however, that there was a striking contrast between the high level of some of these researches and the lack of inspiration and knowledge revealed in some of the applications received for scholarships and grants. Project research, Dr. Witts thought, was always open to the criticism that it encouraged the irresponsible or mercenary applicant; but the fact had to be accepted that project research was likely to be undertaken for some time to come and considerable funds were already earmarked for research on leuchæmia.

Most—but not all—members of the symposium felt that leuchæmia was part of the total problem of cancer, and therefore many cytological and physico-chemical methods of study were common to the whole field. If a hiatus existed in the programme of the symposium, it was the question of the nutritional requirements of the leuchæmic cell and of the possibility of influencing the disease by nutritional means of which some hints had appeared from time to time in the literature. Also, relatively little time had been allotted to the clinical aspects of leuchæmia. Another gap existed in the question of the geographical pathology of the disease. For example, it was said that lymphosarcoma was unduly frequent among the natives of South Africa; this type of finding would be worth confirmation and explanation. A second clinical or demographic problem lay in the evidence that leuchæmia was increasing in frequency in Western Europe and America and changing in the distribution of the various types. This was an aspect of the disease that might well be worthy of sustained and accurate observation.

The symposium opened with a paper on leuchæmia research by J. Engelbreth-Holm, who said that the classification of leuchæmias was even more obscure than it had been ten to twenty years previously, and the relationship between leuchæmias in man and leuchæmias in animals was still very uncertain. In the myeloid leuchæmias difficulties arose as to which attitude to adopt towards myeloid metaplasia, myelosclerosis, and non-leuchæmic myeloid splenomegaly, polysplenomegaly, polycythæmias and leuchæmic erythroblastosis. In animals the tumourous nature of myeloid leuchæmias could not be denied, while in man the question arose whether a tumour and a non-tumour group should be recognized. It was very interesting to know that in fowl, myeloid leuchæmias were excited by the same virus as that which excited erythroblastosis and in some slowly progressing cases brought about myelosclerosis. It was, however, questionable whether one was justified in drawing an analogy between these diseases in fowl and those with similar names in man. Dameshek had put forward the theory that all the

[1] "CIBA Foundation Symposium on Leukæmia Research", edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., assisted by Joan Etherington; 1954. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 312, with 56 illustrations. Price: 30s.

diseases named above were reactions to a myelostimulatory principle. The theory was attractive, but if all these diseases were to be considered as an entity, it was almost impossible to explain the nature of the pathological process. Lymphatic leuchæmia offered difficulties of a different kind, and it was possible that chronic lymphocytic leuchæmia was a metabolic disorder.

As Engelbreth-Holm observed, it is most important to decide on what basis leuchæmias should be classified. Purely morphological and clinical studies are not enough. Obviously it would be of the greatest help to have the actual ætiology of leuchæmias disentangled. To assume a single ætiological agent nowadays is quite unjustified. A good deal of information is being accumulated, but the pattern of it is still very confused. Engelbreth-Holm said that hydrocarbons and radioactivity might be concurrent causes for the development of leuchæmia and that it was possible to include inheritance as a factor. Various nutritional factors had been shown to be important in mouse leuchæmia. Hormonal influences deserved further investigation. Englebreth-Holm stressed the very important fact that almost all therapy of leuchæmias attempted to kill tumour cells; whereas a rational therapy should aim at restoring the abnormal cells to normal conditions. This led directly to the last but not least important field in which future research should be conducted: knowledge of the normal production and differentiation of the cells of the blood was scanty, and little or nothing was known of the process of normal differentiation. One single factor, vitamin B_{12}, was known from research on pernicious anæmia to be fundamental to normal differentiation, not merely of the erythrocytes, but of all marrow cells. There might be other, hitherto unknown, factors which were lacking in leuchæmias.

Joseph H. Burchenal discussed the therapeutic correlation between mouse and human leuchæmia. He said that almost all forms of human leuchæmia could be mimicked by mouse leuchæmia with the possible exception of chronic lymphocytic leuchæmia. Strains of transplanted mouse leuchæmia existed which would respond to every agent effective against clinical leuchæmia. He argued therefore that transplanted mouse leuchæmia was today the best experimental tool for studying the complex problems of the chemotherapy of leuchæmia. M. G. G. Israels, in a paper entitled "The Shortcomings of Animal Research", criticized this argument, and his criticism received much support in a general discussion which followed. It was pointed out that all forms of "treatment" of leuchæmia merely diminished the multiplication of abnormal cells and were therefore symptomatic, not fundamental. Nine physical and chemical agents used in the so-called treatment of leuchæmia were discussed by Burchenal. They were: X-ray treatment; arsenic, benzol and urethane; the alkylating agents such as nitrogen mustard and triethylmelanine; the hormones, ACTH and cortisone; the folic acid antagonists, "Aminopterin" and "Amethopterin", and the antimetabolite 6-mercaptopurine. Israels said it was fair criticism to say that although it was twenty-five years since the transmissibility of mouse leuchæmia had been established, the clinician, battling with the tragedy of human leuchæmia, had derived little benefit from the immense amount of work that had been done, and from the money expended on the study of mouse and fowl leuchæmia. Experimental mouse leuchæmia was now used for "screening" new mitotic poisons, but the original stimulus for the use of such materials had come from direct studies on human subjects. Even the results of screening had to be treated with great reserve. Undoubtedly some important facts had been learnt from the voluminous studies on mouse leuchæmia; but if animal studies were to help the clinician in the future, they would need to be planned in a very different way from those made hitherto. In a later discussion Israels said he would like to see leuchæmia studied in animals other than rodents. The pig was probably from many points of view the animal with a hæmopoietic system nearest to the human. Sir Lionel Whitby said that he was quite sure that the use of antimitotic drugs was the wrong way to treat leuchæmia. As Professor Engelbreth-Holm had said, we know nothing about normal leucocytic growth and the mechanism controlling the release of the cell into the circulation, and such normal activities were not likely to be restored by poisonous drugs. As the result of work in his department he had concluded that the case for the malignant nature of leuchæmia and therefore for much of the utility of the animal experiments was not yet proved. Professor Englebreth-Holm said that perhaps the term leuchæmia was used too freely. The leuchæmias of man which were analogous to the mouse leuchæmias were the autonomous, the frankly tumourous cases. But it was obvious that some of the clinical cases called leuchæmia today were certainly not analogous to mouse leuchæmias.

The very titles of the subsequent papers supported these criticisms of animal work in leuchæmia. Technical studies on "The Method of Quantitative Determination of Leukæmia Virus", on "Endocellular Distribution of the Transmissible Fowl Leukæmia Virus" and on "Transmissible Tumours in Mice" may be of great intrinsic interest, but have little bearing on the problem of human leuchæmia. The laborious "designing" of selective drugs is of interest only if one is convinced that destruction of the leuchæmic cell is the right way to treat leuchæmia. As Engelbreth-Holm commented in a general discussion entitled "Where do we go from here", in the chemotherapeutic field things are well taken care of, but the trouble is that chemotherapy in leuchæmia is of doubtful value. The ingenious suggestion was made that leuchæmias might be classified according to the reaction of the characteristic cell to various cell poisons. Other similar avenues of research were described, but, as De Bruyn pointed out, we still do not know what has gone wrong in the interaction between the leuchæmic cells and their environment. In other words, we still do not know what leuchæmia is.

HEREDITY AND GLAUCOMA.

In recent years there have been many moves by ophthalmologists to diagnose early cases of glaucoma. This has been done by numerous surveys of the general population, chiefly in America, and by the establishment of glaucoma clinics in all parts of the world. The incidence of glaucoma in the general population of patients over forty years is about 1·5%.

L. Kellerman and A. Posner[1] felt that similar surveys could be applied more advantageously to certain selected groups of people. They therefore investigated relations of glaucoma patients. W. H. Hovener[2] has also discussed hereditary aspects of chronic simple glaucoma. Kellerman and Posner examined 192 relatives of patients with glaucoma; eight were found to have definite primary glaucoma all of the chronic simple type. Eleven relatives showed findings which aroused suspicions of glaucoma, but no definite proof of the disease could be obtained. In the great majority of relatives the intraocular tension measured between 20 and 24 millimetres of mercury. This is similar to the average tensions of the general population. Of 359 eyes 61 had tensions of 25 millimetres of mercury or over, and of these 32 were in the discovered or borderline groups, leaving 327 eyes that were considered non-glaucomatous. Among these 327 eyes were 29 eyes (8·9%) with tensions of 25 millimetres of mercury and over. The 45 normal members of the families with discovered and borderline glaucoma account for 11 of these high normal tensions, an incidence of 24·4% as compared with 6·4% in families in which no glaucoma is discovered. The authors were able to confirm R. Tornquist's[3] findings that depth of the anterior chamber is genetically determined and that a shallow anterior chamber is more frequently found among relatives of patients with acute glaucoma than in the population at large.

It is recommended that patients with glaucoma should be advised of its hereditary nature, and in this way relatives may receive the benefit of earlier diagnosis.

[1] *Am. J. Ophth.*, November, 1955.
[2] *Am. J. Ophth.*, December, 1955.
[3] *Acta ophth.*, 1953, Supp. 39.

Abstracts from Medical Literature.

MEDICINE.

Saint's Triad.

E. D. Palmer (*Am. J. Digest. Dis.*, November, 1955) has found that Saint's triad (the coexistence in one patient of *hiatus hernia*, gall-stones and diverticulosis of the colon) appears to be not uncommon. Routine investigation of 170 adult patients with *hiatus hernia* revealed the presence of the triad in 24. The symptomatic results of cholecystectomy were found to be notably poor; but the relief afforded by repair of the hernia was fairly good.

Dangers of Bronchography in Pulmonary Tuberculosis.

K. M. A. Perry (*Brit. J. Tuberc.*, October, 1955) reports two cases in which patients began to expectorate tubercle bacilli after bronchography with iodized oil. In a further three cases tuberculous bronchopneumonia was precipitated by this investigation. The author states that this evidence supports the theory that iodine activates tuberculosis, and it reveals that bronchography is therefore a dangerous investigation in the tuberculous patient.

Coronary Arterial Insufficiency.

A. Vineberg (*J. Thoracic Surg.*, October, 1955) points out that the coronary arteriolar tree is divided into three separate and distinct zones, corresponding to the right coronary artery and the two branches of the left. When coronary arterial disease intervenes, or ischæmia of the heart is promoted through other means, communications between these separate arteriolar zones develop through the formation of collateral vessels. Since coronary arterial disease usually involves the epicardial portions of the major vessels, it is difficult to conceive how the development of collateral vessels between three separate arteriolar zones can bring fresh blood past the blockage, which occurs in the proximal portion of the major coronary arteries. Methods which aim at stimulating the development of intercoronary or collateral circulations are, in his opinion, bound to fail, because they attempt to do what has already been done by Nature. What is necessary in coronary arterial insufficiency is a powerful new source of extracardiac blood, which is brought to the ischæmic heart muscle in such a manner as to bypass the occluded proximal portions of the major coronary vessels.

Atherosclerosis.

M. Israel (*Am. J. Digest. Dis.*, June, 1955) describes an effective therapeutic approach to the control of atherosclerosis, by the prolonged administration of thyroid hormone to patients suffering from coronary disease. These patients were treated with vitamins, to obtain the best oxidative effect, and thyroid (10 to 60 milligrammes per dose). Later, choline hydrochloride 422 milligrammes, inositol 134 milligrammes, and pyridoxine hydrochloride two milligrammes together in tablet form were given one to ten times daily. The dose was assessed empirically, the more atherosclerosis, the more tablets. The latest treatment is that of sodium thyroxin mixed with crystalline vitamin B_{12} in a rather complicated way and named "Co-Thyro-Bal-d". It is injected by the intravenous route twice every ten days or so, and is said to reduce the blood cholesterol content. The first patient treated by these methods improved over five years. A diet of high protein and low fat content was ordered, to keep down the cholesterolæmia. The theory that lack of oxidation in the walls of the blood vessels causes atherosclerosis was adopted. The treatment with thyroid hormone and the oxytropic factors of the vitamin *B* complex (said to be thiamine, riboflavine and niacinamide) and ascorbic acid, was intended to stimulate the metabolism of cholesterol and to suppress hypercholesterolæmia. Records of 20 patients are published, aged from thirty-seven to sixty-six years. The changes in the blood cholesterol content are recorded. The initial reading varied between 174 and 486 milligrammes per 100 millilitres, and subsequent readings revealed a fall of 20 or 30 milligrammes after treatment with "Co-Thyro-Bal-d" in many cases. In others there was a slight increase. The fall in blood cholesterol content seems to have been regarded as evidence of improvement in this report.

Cardiac Disease in Pregnancy.

J. Abramson and B. Tenney (*New England J. Med.*, August 14, 1955) discuss cardiac disease in pregnancy. The incidence of organic heart disease in pregnancy varies from 0·3% to 2%. It is generally accepted that rheumatic heart disease constitutes from 85% to 95% of the cases of organic heart disease in pregnancy, the majority of which are cases of mitral stenosis. Congenital heart disease accounts for 1·5% to 2·5% of the cases of cardiac disease in pregnancy. At present, there is an average death rate of between 2% and 3% in these patients, and congestive cardiac failure is the most common cause of death. The pregnant woman has a profound tendency for salt and water retention, and this action may well be the initial step in the development of congestive heart failure. There are two critical periods during pregnancy and the puerperium when the cardiovascular system is placed under a severe strain; these are at approximately twenty-eight weeks of gestation and in the immediate post-partum period. However, a normal heart has no difficulty in compensating for the increased work. Both the proper appropriate treatment of the heart disease and non-interference with the pregnancy form the present-day approach to this problem. Under correct treatment, the patient should be carried through pregnancy and allowed to deliver herself in the normal manner. The cardiac status should be treated as though the patient were not pregnant, and the obstetric problem as though she did not have heart disease. Failure to make a proper diagnosis and to evaluate the cardiac status may threaten the life of the patient. Hospitalization at the first sign of decompensation, and continued hospitalization until well after delivery are essential. Most authorities agree that interference with pregnancy during advanced failure can result in disaster. In mitral stenosis, resort may be had to valvuloplasty, but this approach should be avoided during the pregnant state unless it appears necessary to save life. Frequent observation of the patient is most essential during labour and delivery and in the post-partum period. Treatment of cardiac emergencies with a minimum of delay may make the difference between life and death. Vigorous treatment of pulmonary œdema must be pursued. Fluid administration in the presence of heart disease should be slow and in minimal amounts during pregnancy and delivery. It is common practice to sedate patients heavily during labour with barbiturates and opiates. Some clinics prefer inhalation anæsthesia, and many others prefer regional types of anæsthesia. It is thought, however, that the choice of a trained anæsthetist is more important than the method employed. Cæsarean section is to be recommended only for obstetrical reasons. The use of oxytocics in the third stage of labour should be avoided unless it is necessary for uterine atony.

Pleural Lymphatic Dilatation.

B. Levin (*Am. Heart J.*, April, 1955) draws attention to certain transverse linear streaks in the lower halves of the lung fields in postero-anterior X-ray films of the chest, usually bilateral, some two or three centimetres long, arranged in parallel fashion, one above the other and extending outward to the pleural surfaces. These striæ are sometimes seen in the presence of silicosis and have been attributed to dilated intercommunicating pleural lymphatics. The author shows that they are due to the same cause as when they are seen in the films of patients with mitral stenosis. Their presence indicates the existence of chronic pulmonary venous and arterial hypertension.

Patent Ductus Arteriosus.

I. J. Pinto (*Am. Heart J.*, July, 1955) discusses the various auscultatory findings in patients with patency of the *ductus arteriosus*. He explains that these findings are dependent on the pulmonary arterial pressure and the volume of flow across the shunt. From auscultation, therefore, may be obtained an idea of the size of the ductus and of the degree of urgency of its operative closure. When there is a typical continuous murmur at the pulmonary area but no diastolic murmur at the apex, the shunt across the ductus occurs both in systole and in diastole, but the volume of flow across the shunt is not sufficient to cause an apical diastolic murmur; therefore the ductus is small, and operative closure is not urgently necessary but is a safeguard against bacterial endocarditis. When auscultation reveals both a typical continuous murmur at the pulmonary area and a diastolic murmur at the apex, there is a large volume of shunt, and early operative closure is indicated. When there is a typical pulmonary murmur at the area without a diastolic murmur at the apex, pulmonary hypertension is present, and

early surgical correction is necessary. When there is an atypical murmur at the pulmonary area with a diastolic murmur at the apex, the ductus is of large size, possibly larger than the pulmonary artery and aorta, and should be closed.

Myocardial Infarction in Women.

T. N. JAMES, H. W. POST AND F. J. SMITH (*Ann. Int. Med.*, July, 1955) review 146 cases of myocardial infarction in women. The average age of these women at menopause was 47·4 years, and the average interval before the subsequent infarction was 16·6 years. The incidence of infarction rose sharply after the fifth decade. Hypertension (blood pressure greater than 160 millimetres of mercury, systolic, and 100 millimetres, diastolic) and/or diabetes was present in 69·9% of the entire series, 63·6% of the living patients, 77·6% of the patients who died, and 90·9% of the premenopausal group of women. In this latter group only one of 11 women did not have other diseases which predisposed to coronary disease.

Pulmonary Disease and Zenker's Diverticulum.

L. E. HAWES AND J. H. WALKER (*New England J. Med.*, 253: 209, 1955) describe two patients in whom Zenker's diverticulum (pharyngeal pouch) led to inflammatory disease of the lung and was repeatedly overlooked. One patient had a localized area of lipoid pneumonia in the lower lobe of the right lung, while a second patient had multiple abscesses in the upper and, later, the lower lobes of the left lung. The authors emphasize the opinion that before any operation on a pulmonary lesion of unknown ætiology is undertaken, the absence of primary œsophageal disease should be proved by barium bolus X-ray examination.

"Ambonestyl."

B. B. CLARK AND B. ETSTEIN (*New England J. Med.*, 253: 217, 1955) report their preliminary observations on the use of "Ambonestyl", a new anti-arrhythmic compound, in man. They believe that it may have some advantages over both procaine amide and quinidine in the treatment of ventricular arrhythmias because of the absence, when "Ambonestyl" is used, both of depression of cardiac conduction and of the lack of serious hypotension on its administration by the intravenous route.

Chlorpromazine and Rauwolfia in Essential Hypertension.

H. B. EIBER (*J.A.M.A.*, July 2, 1955) presents a preliminary report on combined treatment with chlorpromazine and *Rauwolfia serpentina* in essential hypertension, based on a series of 75 cases. A capsule containing 15 milligrammes of chlorpromazine and 50 milligrammes of *Rauwolfia serpentina* (whole root) was given three times daily to each of the patients, all of whom had hypertension, ranging in degree from mild to severe, and classified as follows: mild with blood pressure above 150 millimetres of mercury, systolic, and 50 millimetres, diastolic, with mild complaints; moderate with blood pressure above 200 millimetres of mercury, systolic, and 100 millimetres, diastolic, with more severe complaints; severe with blood pressure higher than these figures and with severe complaints and with the patient confined to bed. During treatment, blood pressure readings were obtained with the same instrument, by the same observer, and in the same office or at the bedside. No other medication was prescribed for the patients during the course of treatment, and they were divided into three groups: those given chlorpromazine alone, followed by the combination; those given rauwolfia alone, followed by the combination; those given the combination of the two preparations, either preceded or followed by a placebo. The patients were observed at weekly intervals. More than 85% of those with mild hypertension had demonstrable, significant lowering of the blood pressure; more than 70% of the patients with moderate hypertension were benefited; and more than 60% of the patients with severe hypertension were improved. Of the whole series of patients 95% volunteered the information that they felt considerably improved and had less anxiety regarding their cardiovascular status and less headache. In most cases maximal hypotensive response was observed in two or three weeks. Only one patient was unable to tolerate the preparation because of marked somnolence. The blood pressure readings of almost all the patients who were given placebos during the course of therapy increased to original levels after ten to fourteen days of placebo therapy. When combined therapy of chlorpromazine and rauwolfia was reinstituted, improvement was again observed after two weeks. No cases of postural hypotension were noted either during or after treatment, and bradycardia was not significantly greater than when the patients were treated with *Rauwolfia serpentina* alone. The improvement with combined chlorpromazine and rauwolfia therapy was much greater than with the use of either preparation given alone. No toxic action of these drugs was noted in the series. The duration of hypotension was approximately six to eight hours with each dose, and there were no side effects. In an addendum, the author states that a further group of patients have been treated, making a total of 350 with hypertension, and that results are even more promising with a dosage schedule of 25 milligrammes of chlorpromazine combined with 50 milligrammes of *Rauwolfia serpentina* (whole root) given by mouth three to four times daily. After eighteen months of therapy, no jaundice or other side effects were observed in any of the 350 patients.

Jaundice Occurring During the Administration of Chlorpromazine.

C. H. STACEY, J. G. HOWLETT AND M. M. HOFFMAN (*Canad. M.A.J.*, September 1, 1955) discuss the finding of jaundice occurring during the administration of chlorpromazine. Their observations are based upon a study of 170 patients treated with chlorpromazine for mental illness, eight of whom developed hepatitis with jaundice. Seven additional patients of the series were observed to have an elevation of the serum alkaline phosphatase level as the sole manifestation of liver damage. Jaundice appeared between the sixteenth and twenty-first days following the onset of treatment, and there was no correlation with sex, age or previous state of health, or with the dosage of chlorpromazine. The symptoms at onset were those of acute gastro-enteritis with anorexia, nausea, vomiting and the passage of loose stools dominating the clinical picture. Jaundice occurred several days after the onset of these symptoms. Liver biopsy in one case revealed histological features of progressive intralobular biliary stasis, with non-suppurative cholangiolitis. There was no significant change in the parenchymal cells. Hepatomegaly and pruritus were common discoveries, and the biochemical findings were characterized by hypercholesterolæmia and negative sero-flocculation reactions. The authors were impressed by the alteration in the serum alkaline phosphatase determinations in those patients who developed jaundice. Because of these observations a plan was devised of assessing patients who were receiving chlorpromazine. The programme consisted of serial determinations of the alkaline phosphatase level at regular intervals varying from three times a week to weekly. Whenever the serum alkaline phosphatase level had a significant elevation, chlorpromazine was discontinued. In these patients, subsequent determinations of the alkaline phosphatase level revealed a fall towards initial values. The therapy followed was that in use generally for infectious hepatitis. Except for one death as a complication of liver biopsy, all the patients eventually recovered. The authors conclude that if the drug is discontinued when the alkaline phosphatase level is first found to be elevated, further disturbance in hepatic function can be avoided.

Potassium, Heat Exhaustion and Fibrocystic Disease.

S. J. HARRIS AND R. L. LESTER (*New England J. Med.*, 253: 233, 1559) report two cases of fibrocystic disease complicated by heat exhaustion. They point out the importance of potassium deficiency in the genesis of the hypochlorœmic alkalosis characteristic of fibrocystic disease and stress the necessity for adequate parenteral administration of potassium in treatment. They also suggest the administration by mouth of potassium, in addition to salt, in prophylaxis.

Lupus Erythematosus.

G. C. MEACHAM AND A. S. WEISBERGER (*Ann. Int. Med.*, July, 1955) review their experience with 25 cases of disseminated *lupus erythematosus*. They emphasize the marked variations in the course and clinical picture of the disease. In their most recent series, five patients had thrombocytopenic purpura, four had hæmolytic anæmia, two had circulating anticoagulants, seven gave false positive serological reactions for syphilis, and five had Raynaud's phenomenon. One patient developed intestinal perforation. All these individual features may be the initial manifestation of the disease, and they may occur before other less uncommon features. The authors place great reliance on the specificity of the *lupus erythematosus* cell.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on July 19, 1955, the medical superintendent, DR. NORMAN H. ROSE, in the chair. The principal speaker was DR. F. L. RITCHIE.

Clinical History.

The following clinical history was presented.

A young woman, twenty-four years of age, had been ill for nine months or so before coming to hospital. During that time her menstrual periods had stopped, her abdomen had swollen, she had had several attacks of diarrhœa, each lasting three or four days, she had lost a great deal of weight and she had become increasingly breathless after moderate exertion. Micturition was frequent and often painful, causing pain to pass from the right loin to the right groin. Three days before her admission to hospital her abdomen had suddenly swollen further; the swelling had also affected her legs, and she had become breathless even at rest. There was no record of her family history or of any past illnesses, except that she had had an operation one year previously for what was thought to be gall-bladder trouble, but the viscus was not removed or drained.

On examination she was found to be very thin with œdema of the legs, thighs and back up to the lumbar region, and ascites. The chest was dull at its bases. The apex beat of the heart was six inches from the mid-line in the fourth intercostal space. The heart sounds were normal: no murmurs were heard. The blood pressure was 130 millimetres of mercury, systolic, and 95 millimetres, diastolic, and the pulse had a rate of 110 per minute, and was regular in time and amplitude. No venous congestion was apparent. The abdomen was grossly distended. No organs could be palpated. Superficial veins could be seen, but they were not prominent. No albumin or sugar was detected in the urine.

Investigations and Progress.

Paracentesis yielded 16 pints of clear fluid, which contained 1·9 grammes of protein per 100 millilitres and had a specific gravity of 1·013. Microscopic examination showed 1200 red blood cells, 100 endothelial cells and 10 lymphocytes per cubic millimetre; no organisms were grown. An enlarged, hard, nodular liver could then be felt.

The blood had a hæmoglobin value of 10·9 grammes per 100 millilitres, with moderate macrocytosis. The white cell count was 17,300 per cubic millimetre with 76% of neutrophile cells, showing some toxic degeneration, 11% of lymphocytes and 13% of monocytes. Platelets were plentiful. The sedimentation rate was four millimetres in one hour. The serum protein content was 5·3 (albumin 3·3, globulin 2·0) grammes per 100 millilitres. The bilirubin content was within normal limits.

X-ray examination of the chest showed an elevated diaphragm on both sides, causing compression of the lung bases, with no changes in the upper parts of the lung fields. The cardiac outline was not enlarged.

On X-ray examination of the renal tract, no calculus was visible in a plain film. In an excretion pyelogram both kidneys functioned; the left appeared normal, but the right had enlarged calyces and pelvis, suggesting a partial obstruction of the ureter below the pelvic brim.

An electrocardiogram showed myocardial degeneration.

Gynæcological examination under anæsthesia revealed no abnormalities. A surgeon considered malignancy to be the most likely diagnosis and advised against any operative procedures.

The patient's temperature occasionally rose to 99° F. *Paracentesis abdominis* was repeated several times. Her condition gradually deteriorated, and she died two months after admission to hospital.

Clinical Discussion.

DR. NORMAN H. ROSE: I think those of you who have read the case will be frightened by its simplicity, for there appear to be very few hooks on which the speaker can hang his hat. I introduce Dr. Ritchie.

DR. F. L. RITCHIE: Thank you, Dr. Rose, ladies and gentlemen. There is one other clue which I have and you have not. I was warned, when asked to talk about this case, that a number of pathologists would be here today to talk about this case, and that they would want to talk at some length themselves. With this brightening piece of extra information, I proceed.

First of all, when you review this young woman's symptoms the earliest clue is that one year ago a surgeon presumed, evidently from what she told him, that she had gall-bladder disease. So I think it is fair to assume that she probably had some pain and was probably tender in her right hypochondrium, and that her liver, if palpable, was not, as subsequently found during examination, craggy and hard. However, he did operate on her, and I presume because of what he saw he decided that he would neither remove nor drain that organ. I imagine that he saw something in the liver which has subsequently progressed and created the remainder of her clinical picture. It is unfortunate that we know nothing about what led her to consult a surgeon a year ago. The first we know of her illness, as you read, was when she presented with abdominal swelling, considerable weight loss and some disturbance of urination.

One can tackle this problem in a variety of ways. I thought I would tackle it in a clinical fashion and talk about causes of this picture which could occur primarily outside the abdominal cavity, and then causes which could occur within it.

There is only one pattern outside the abdominal cavity which can mimic this picture, and that is heart failure and constrictive pericarditis. As you know, both those illnesses are prone to cause ascites and enlarged liver, œdema and breathlessness. But there are several pieces of information here which rule out these possibilities. Though the resident medical officer found her heart to be enlarged clinically—six inches out (was it not?) in the fourth intercostal space—that finding was not confirmed radiographically. She did not have an enlarged heart. The second important matter, particularly important, is the statement that during her physical examination there was no venous congestion. Now, it is quite impossible to have ascites from cardiac failure without a raised venous pressure. A considerable rise would be found also in constrictive pericarditis. So these possibilities are ruled out. Finally, it is very uncommon for heart failure to cause gross enlargement of the liver, and even then it is seldom nodular, as this one apparently was.

Now, of the primary abdominal causes let us consider some which actually arise in the liver. One of the first, perhaps to be dismissed, is primary hepatoma. As most of you are aware, primary hepatoma of the liver occurs most commonly in what is already a cirrhotic liver, therefore more commonly in men, and usually in a later age group than this. It is characterized by rather vague gastro-intestinal symptoms, often with a good deal of pain in the right hypochondrium because of associated perihepatitis. Other features are rapid weight loss, fever and leucocytosis, and there is a rapidly recurring ascites. Many hepatomas, as you know, metastasize not only to other parts of the abdominal cavity, but also to the lungs, and hæmoptysis is therefore common. As you will see she had some of the characteristics which would suit a hepatoma. I have seen only very few, I think two or three, and they have all been single, large tumours in cirrhotic livers. However, multiple primary neoplasms can occur and produce a hard, nodular liver. I find there is no certain way from this history of completely ruling out this latter prospect. But I think it is unlikely, as there is no evidence here that the girl has had a cirrhotic liver for some time past, and it is unusual for hepatoma to occur in the young, particularly the young non-cirrhotic.

When thinking of other primary hepatic conditions one is straightway confronted with a confusion of classification. I have selected Himsworth's classification, perhaps known to most of you, from his Lowell Lectures of 1947, given in Boston. He defines two types of chronic liver disease. The first is chronic massive hepatitis which occurs from post-necrotic scarring. The nature of this is the early appearance of clear evidence of liver failure and the late appearance of portal hypertension with enlargement of the spleen and gross nodularity of the liver. In this girl there is no evidence that she had portal hypertension. She had ascites but no superficial veins of any prominence on her abdomen, and no history of hæmatemesis or bleeding piles. I have attributed her ascites to local peritoneal irritation rather than portal hypertension. I feel therefore that this particular type of primary liver disease is also unlikely.

The diffuse type of hepatic fibrosis, the alcoholic cirrhosis we usually think of (really recurrent zonal necrosis followed by fibrosis, with which alcohol is often associated), is very slow to develop and usually presents with evidence of portal

hypertension, though the patient otherwise feels quite well. Jaundice is slow to appear, and the whole pattern of the illness usually continues on over a number of years, perhaps ten or fifteen, culminating in jaundice and final liver failure towards the end. Again this does not fit the pattern presented by this girl. Diffuse hepatic fibrosis may also occur from various infiltrates, of which there are but two which one ought to mention. One is syphilis. Single or multiple gummata occur in tertiary syphilis of the liver, and when this is so, portal hypertension is a rare associate, the course is usually slow and leucopenia is the rule. This girl had a marked leucocytosis. There is no evidence elsewhere that she had syphilis. The other type of infiltration concerns the reticuloses, of which Hodgkin's disease and lymphosarcoma are the two occurring most readily to mind. There is no evidence from her examination that she had glandular enlargements elsewhere, and it seems unlikely that she would have Hodgkin's disease perpetually confined to the abdominal cavity. A retroperitoneal lymphosarcoma or sarcoma can, of course, be much more localized. It is within the bounds of possibility that her liver had become infiltrated with lymphosarcomatous deposit, which had also, perhaps, obstructed her inferior *vena cava* and caused ascites with peripheral œdema. I have no way, again, of knowing whether that had happened, but I feel that it is unlikely.

The other primary condition occurring in the liver, which I feel in this instance does not operate, is biliary cirrhosis. There are two varieties. One, originally described by Charcot, is caused by obstruction of the biliary tract—particularly by common duct stones and such like or extra-duct neoplasia. The predominant picture is one of prolonged obstructive jaundice associated with fever, liver enlargement and, of course, unless recognized early, ending fairly rapidly with death. This girl apparently was not jaundiced, and there is no other evidence that she had obstruction to her biliary outflow. The second type of biliary cirrhosis, described by Hanot, of which I do not think I have ever seen a case, is a non-obstructive type. Patterned by intermittent jaundice with increase and decrease in liver size one finds a smooth liver enlargement. Ascites is common. Most feel this picture represents a low-grade infection caused either by *Bacillus coli* or by *Streptococcus viridans*. There is nothing here to warrant discussing either of those further.

We are left then with a number of conditions which are primarily extrahepatic in origin, but which, in fact, produce predominantly liver effects. Of that group, carcinoma with secondary spread to the liver is the most common. Secondary carcinoma of the liver may, of course, arise from a primary in any organ of the body. Usually the initial site is intra-abdominal or pelvic. Such may occur at any age depending on the age groups of carcinoma. There are a number of characteristics of secondary carcinoma of the liver which, to me, fit this girl's picture better than any of the other possibilities. The first is that she was wasted. With primary carcinoma, particularly of the bowel, and with secondary deposits in the liver, wasting is a very early and a very marked feature. The second is that patients with secondary carcinoma of the liver very commonly run periodic fevers, sometimes low-grade, sometimes of quite a septic variety. This girl had apparently had some fever. Secondary carcinoma of the liver frequently shows a clear-cut leucocytosis, as though one were dealing with a pyogenic infection; this, too, she showed. This leucocytosis is usually associated with a relatively high blood sedimentation rate, which she had not. I find it hard, whatever diagnosis I make, to reconcile the blood sedimentation rate of four millimetres per hour with a leucocytosis of 17,000 per cubic millimetre. Should I be wrong, as well I might be, it will be interesting to know what explanation will fit that apparent inconsistency. The other feature of secondary carcinoma of the liver which, I think, is important is that pain is very frequently felt in the hypochondrium, not only a dull, dragging pain, but also sharp episodes of pain. When I was imagining this case some weeks ago and trying to put myself as the original doctor who saw her, I imagined that if she had had sharp episodes of hypochondrial pain, a surgeon could have been bold enough to mistake that for gall-bladder disease, and even bold enough to open her up—which, in fact, he did.

In secondary carcinoma of the liver, the spleen is uncommonly enlarged: when it is so, it is because there is an associated portal hypertension due to actual secondary spread into the portal system. Whether or not there is jaundice depends entirely on the chance deposition of the secondary deposits.

There is one portion of the history which I have quite failed to bring to your attention yet. Her "micturition was frequent and often painful, causing pain to pass from the right loin to the right groin". Then when you look at the intravenous pyelogram there is some evidence that there might have been obstruction on the right side in the lower part of the ureter. Can we link all this urinary disturbance with a secondary carcinoma? I think if one wanted, one could imagine that she had a primary carcinoma of the

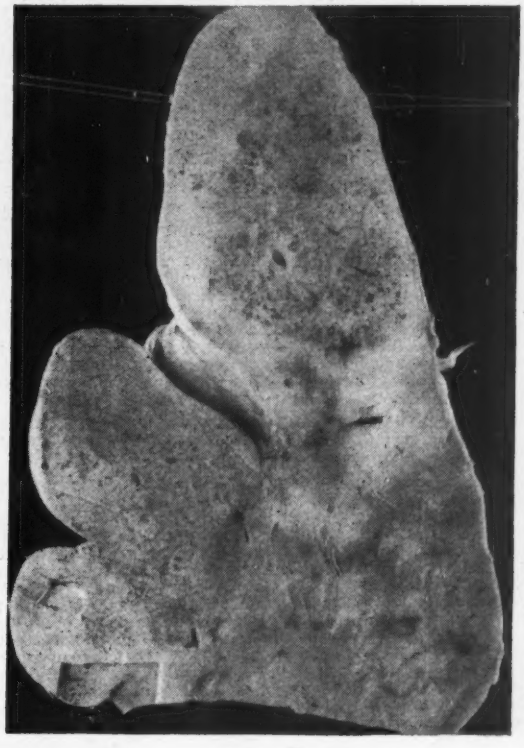

FIGURE V.
Liver.

ovary with some spread back to where the ureter runs over the brim of the pelvis, and likewise some spread from the ovary to the liver. Another primary site could be the colon, and, as many of you know, carcinoma of the colon tends very often to metastasize to ovary as well as to liver. I feel it is idle to guess where the primary carcinoma was, if in fact this was carcinoma at all, but I would think it was somewhere in or near the pelvic brim and secondarily scattered through the peritoneum to cause irritation and ascites.

Therefore, just to summarize my feelings about this, I feel that she probably had a carcinoma with secondary spread to her liver. I am not by any means sure that she had not a primary hepatoma, but I think it is statistically less likely. There is no way I can find from this history of separating it out. I feel that the other matters I have mentioned, like primary hepatic disease, are, on the whole, far less likely.

DR. ROSE: Dr. Ritchie has committed himself about a difficult case. This meeting is both clinical and pathological. There are going to be several contributions by pathologists, so could we have some preliminary discussion by the clinicians?

DR. W. L. CALOV: When I read through this case, I noticed that the patient's history started twelve months ago. I do not know whether the surgeon saw anything wrong with the liver or not, but I think it may well be that he saw nothing wrong with the liver. My first idea was that there was a primary disease of the liver, but Dr. Ritchie has made out such a remarkably good case against this that I am

wavering considerably. But I suppose I had better stick to my first idea and say that she had primary disease of the liver, and, I think, it is certainly neoplastic. I will be tremendously surprised if it is not a carcinoma of some kind. Whether or not there is something lower down with secondaries scattered throughout the liver, I do not know. The other symptoms, such as painful micturition, might be incidental. By the way, I notice that the gynæcologist examined her under an anæsthetic. Therefore, I assume she was a single woman, which rules out some of the possibilities in the pelvis which would have to be considered if she were married. I really think this is carcinoma, and my first idea was primary carcinoma of the liver. I had better stick to that.

Dr. J. N. Sevier: I must congratulate Dr. Ritchie on the very thorough and competent way he attacked this, and I feel that he is right. One can hardly go against his conclusions. There are just a couple of points which struck me. One is this sedimentation rate of four millimetres in one hour. No matter what the cause of her condition, I cannot understand that: possibly a typographical or technical error. The other point which struck me was the 13% monocytes of the 17,300 white cells. Whether or not that is significant I would not know, but one thought that occurred to me was: might this be a case of tuberculous peritonitis? They have been confused with malignancy ere now. There is one in the ward at the moment, a Chinese girl, a woman of thirty-nine, with a story not unlike this. Menstrual symptoms often may occur and be prominent, urinary symptoms occur, and I have seen them before written off as malignancy and left to perish when autopsy revealed the diagnosis. In this day and age it is amenable to treatment. I do not know when this patient was admitted. I agree with Dr. Ritchie, but I would keep that as a "saver" up my sleeve—tuberculous peritonitis.

Dr. Rose: Dr. Robertson, there have been some queries asked about certain hæmatological anomalies here. Would you care to comment on these?

Dr. T. I. Robertson: I do not think I have much constructive to add. There is an obvious leucocytosis, and I think it is very difficult to explain a normal sedimentation rate, irrespective of what the disease may be, in the face of a leucocytosis. My own feeling would be to have the test repeated. We must ask Dr. Whyte and Dr. Johnston their opinion because they have both done work on erythrocyte sedimentation. Dr. Sevier's indirect query about the monocytes I think might have referred to the suggestion that monocytosis can occur in tuberculosis. Well, it is recorded, but it is not a very good indication. The moderate macrocytosis is another point, and this occurs commonly in chronic liver disease. The explanation of that is not surely known; it does not appear to be related to megaloblastic maturation because the marrow of these people shows normoblastic maturation.

Dr. Rose: Have you any comments, Dr. Johnston?

Dr. G. A. W. Johnston: Only to say that I think the sedimentation rate is correct, and I have an explanation for it which I could perhaps give later.

Dr. R. J. Walsh: I wondered whether Dr. Ritchie could explain the attacks of diarrhœa this woman had.

Dr. Phyllis Anderson: Mr. Chairman, I should like to say a word or two in this age when infectious diseases are not fashionable. It seems to me that the history of onset and the irregular course of the illness might possibly be explained by an original attack of infectious hepatitis leading to massive fibrosis of the liver. I think that such an initial onset could contribute to the explanation of the diarrhœa in the original illness, and it might contribute to the explanation of the ascites. I am impressed and caused to wonder at Dr. Ritchie's suggestion that 16 pints of fluid could result from perihepatic irritation: it seems a little large to me. The only suggestion I am putting forward is that a chronic infectious process, a chronic fibrotic process, superimposed on an original acute infectious hepatitis, might be considered. I am not putting any money on it.

Dr. J. Schneiweiss: Might we at this stage ask what the result of liver function tests, if any, were?

Dr. Rose: Have we that information?

Medical Officer: Everything that is in the records is before you.

Dr. Rose: While the screen is being adjusted and before Dr. Palmer gives the autopsy findings, Dr. Ritchie has some questions to answer.

Dr. Ritchie: With regard to Dr. Walsh, in chronic liver disease from almost any cause, I think I am right in saying there may be periodic diarrhœa. It had no specific meaning to me. Dr. Johnston, you frightened me terrifically! If that sedimentation rate is normal in the face of that leucocytosis, then my answer must be wrong. Anyway, I stick to it. Dr. Anderson, I agree with your comment. I feel, in explanation against it, that if this were chronic fibrotic liver disease of the massive fibrosis type which I mentioned, I would expect there to be more clear-cut evidence of portal hypertension. I am not saying you are not right.

Autopsy Report.

Dr. A. A. Palmer presented the following autopsy report:

The body was that of a young women showing extreme emaciation. The body weighed 94 pounds. Numerous petechial hæmorrhages were present over many parts of the body, particularly the skin of the abdomen.

Thorax, thymus and mediastinum: These were normal.

Pleural cavities: An abnormal amount of fluid was present in the pleural cavities of each side.

Lungs: The pleural surface of each lung was studded with small hard nodules of cartilaginous consistence, one to three millimetres in diameter. The cut surfaces of the lungs were congested but normal in pattern.

Heart: The heart weighed five and a half ounces (normal range eight and a half to ten ounces). The pericardial sac contained about four ounces of straw-coloured fluid. The heart showed an extreme degree of brown atrophy, but there was no abnormality of the chambers, orifices or coronary arteries.

Abdomen and peritoneum: The peritoneal cavity contained approximately 10 pints of straw-coloured fluid in several loculated cavities. No mesenteric or omental fat was seen, but scattered throughout the whole of the peritoneal surface were small, white, almost cartilaginous nodules ranging from one to five millimetres in diameter. These nodules were present also in the omentum.

Liver: The liver weighed 55 ounces (normal range 50 to 60 ounces). The surface of the liver was white and lobulated, and numerous fine dilated vessels were present in the serosa. The cut surface of the liver showed a replacement of the parenchyma by hard, grey tissue with irregular reddish-brown mottling sparsely scattered throughout. Two hypertrophied accessory lobes, one measuring about five centimetres and the other four centimetres in diameter, were almost the only liver tissue presenting a normal parenchymal pattern.

Spleen: The spleen weighed three ounces (normal range five to seven ounces). The spleen was small and firm, but otherwise normal.

Kidneys: The right kidney weighed six ounces and the left kidney weighed four and three-quarter ounces (normal range five to six ounces). Both were normal.

Œsophagus, stomach and intestines: These were normal.

Pancreas and suprarenals: These were normal.

Pelvis, bladder and genital organs: Apart from the above-mentioned peritoneal nodules, the pelvic organs were normal.

Brain and cord: These weighed 40 ounces. They were normal.

Pathological Discussion.

Dr. Palmer: Dr. Hirst, would you like to open the discussion?

Dr. E. Hirst: It is true that I reported on these sections some years ago. I would like to tell you about Dr. Hertzler, who was a surgeon as well as a pathologist, and who has written a series of monographs on surgical pathology. On one occasion I sought his aid and found the following advice. When faced with a difficult diagnosis it was his practice to put the sections aside for five years. At the end of this time, if the patient was alive and free of the disease, he regarded it as benign, and if the patient had died of the disease, it was malignant. He added that not all pathologists found this convenient, especially if the surgeon was pressing them for a diagnosis. In that case it takes a person of only limited experience to compose a report which in the course of a page will utterly confuse the surgeon; and with increasing experience similar confusion can be created in a quarter of a page.

When I look back on my lengthy report after some years, I realize I had better say that I still do not know what the diagnosis is. However, on reviewing the sections certain features are notable. I am satisfied that histologically and biologically the disease is a malignant newgrowth.

Is it then a growth with multiple primaries in liver, lung, peritoneum, lymph nodes and ovary, such as Hodgkin's disease, reticulosarcoma or even Letterer Siwe's disease, which occurs rarely in the adult? Without enumerating the reasons I discard these possibilities.

It must then be carcinoma, sarcoma or teratoma. Teratoma occurs in the liver in children and enlarges it. Certain types of sarcoma have from a histological point of view much in common with the lesions in this case. For example, Dr. Palmer has demonstrated the cartilage-like nodules containing cells with abundant cytoplasm resembling strap cells. In two situations, ovary and peritoneum, there are benign-appearing glandular structures. These appearances are found in mixed mesodermal tumour, and the histological appearances make this diagnosis most attractive. However, I have been unable to find cross striations in the strap cells, and on critical examination I have concluded that the glandular structures found belong on the one hand to the ovary and on the other are serosal cells forming pseudoacini after incorporation by the growth. Furthermore, the biological pattern of the growth does not resemble that of Müllerian mixed tumours, and for these reasons the diagnosis of mixed mesodermal tumour cannot be sustained. For similar reasons neurogenic tumours and fibrosarcoma have been discarded as possible diagnoses.

This leaves carcinoma, and the problem arises of indentification of the primary growth. Some carcinomas, for example, of skin and kidney, mimic sarcoma. However, we have in this case, in addition to histological evidence, the post-mortem findings indicating clearly the biological behaviour of the growth, and this is to my mind very strong evidence of its nature.

I suggest, therefore, that the diagnosis is metastatic carcinoma arising from an occult primary. I will not weary you by listing hidden primaries, but will say that on flimsy evidence the primary lesion might have been in the breast.

I have found a report[1] of a woman of twenty-eight years who, seven years after the removal of a carcinoma of the breast, was found *post mortem* to have a small shrunken liver, which, as in this patient, appeared so much like cirrhosis that the authors called it secondary atrophic carcinomatous cirrhosis of the liver; and that is what I suggest is the diagnosis in this patient.

DR. PALMER: Thank you very much, Dr. Hirst. Dr. ten Seldam has an interesting contribution to make.

DR. R. E. J. TEN SELDAM: Thank you, Dr. Palmer, for giving me the opportunity to say a few words. I am afraid I cannot agree with Dr. Hirst. I do not think this is a carcinoma but a malignant tumour of mesenchymal origin—in other words, some kind of sarcoma. Just before the war I received surgical material of a similar case on which autopsy was refused. The material consisted of the appendix, the right ovary and biopsies of liver and omentum. The histology was identical with the case Dr. Palmer just showed us. My case occurred in a woman of twenty, also with extreme wasting and with extensive calcification in the liver. Calcification of tumours in the liver is hardly known, and to my knowledge was not reported till 1941, when I reported my case in collaboration with the physician Dr. Zuidema. As there was also extreme necrosis in the liver, just as in this case, I considered the calcification to be secondary to this necrosis in what probably was a comparatively slow-growing tumour.

I found the extreme wasting in my case difficult to explain, and as this is also a remarkable feature of Dr. Palmer's case, I wonder if some general disturbance of endocrinological origin may not be a part of the condition. The considerable increase in basophile cells in the pituitary may point in that direction also.

I was fortunate enough to be able to make fat stains, but could not demonstrate any inside the actual tumour cells. Neither did the vacuoles contain glycogen or mucin. A faint pink stain was present in the stromal background. As myxomatous degeneration can take place in many tumours of mesenchymal origin, this finding cannot be interpreted as being of great importance.

Even so, I reported my case as a peculiar form of myxoma for lack of a better suggestion. I had my doubts at that time about the correctness of that diagnosis, and I feel convinced now that it was wrong. I have no alternate suggestion to make, but I still think it is a malignant tumour of mesenchymal origin.

DR. PALMER: Thank you. The X-ray appearance in your case was extraordinary because the entire liver was absolutely opaque in the ordinary X-ray film. We found no calcification in this case. We found exactly the same staining of the background, with stains for mucin and absence of staining of the vacuoles, as in your case. The wasting in this case was really something quite unusual, more than one usually finds in malignant disease. I think from what I have heard of your description that whatever was the answer in one case is also the answer in the other. Professor Magarey, would you care to comment?

PROFESSOR F. R. MAGAREY: I feel that, following the last two speakers, I should apologize for never having seen anything like this case before. At least I can approach it with an unbiased mind. I think we are dealing with a neoplasm, probably a sarcoma. I also think it is rather more than coincidence that some of the nodules described are cartilaginous in consistency, because, particularly in some of the small ones found in the lung, there is a very strong tendency to mimic cartilage histologically. The long, pink-staining cells could, perhaps, be of muscle origin. It is said that they do not stain for myofibrils, so we are no further forward. I think it is a sarcoma which has metastasized, from an unknown primary site, and I cannot put a name to the tumour. It is undifferentiated, but attempting to produce fibrous tissue and, amongst other things, cartilage. There is one other point. There may be something else going on in the liver as well. I am not sure whether there is not, in fact, a healed massive fibrosis. Or perhaps there are multiple organized thrombi on the portal veins, which you have so ably demonstrated, with the resulting healed fibrotic infarcts. I know that this is not supposed to occur in the liver, but perhaps it has done so in this case. I am certainly not convinced that all of the liver which we can see, both with naked eye and microscopically, is part of the tumour. In other words, I am prepared to believe that there is some chronic hepatic disease in its own right as well as the neoplasm.

DR. PALMER: I think some of the tissue in those veins in the liver is very like the tumour nodules. My impression is that there are tumour emboli in the branches of the portal vein, and perhaps by blocking these the emboli have caused secondary fibrosis in the affected parts of the liver.

PROFESSOR MAGAREY: I am quite willing to accept that explanation.

DR. PALMER: Dr. Tebbutt, would you like to give your opinion?

DR. A. H. TEBBUTT: Well, I had a look at these sections and was frightened to make a diagnosis. After hearing this discussion I am even more frightened to do so. I could, of course, take a free kick at it, but I would prefer to hand the ball over to Professor Inglis. I think the older physicians would have called this polyorrhymenitis or polyserositis. But if you look up the older text-books on those topics, you will find that in most of them no real diagnosis was made. This is very like the present case. The suggestions made by Dr. ten Seldam and Professor Magarey are possibilities, but I do not feel convinced that they are correct. I do not feel convinced that it was neoplastic.

DR. PALMER: Thank you. Dr. Shearman, would you like to contribute?

DR. C. H. SHEARMAN: I cannot add anything worth while. If Dr. Tebbutt was frightened to make a diagnosis, I definitely could not make one. I did think some of those large cells reminded me of primary hepatic tumour, and I thought there might have been such a primary, which had been strangled by all the fibrosis, such as one sees sometimes in scirrhous cancer of the breast; but I could not correlate the other peculiar connective tissue changes which were spread over parts of the omentum. The nearest I could get to it was that it may have been a primary hepatic tumour.

DR. PALMER: Thank you. Professor Inglis?

PROFESSOR W. K. INGLIS: The microscopic changes in the liver are difficult to interpret; they vary in different parts. In some situations there is overgrowth of fibrous tissue, in others the structure strongly suggests malignancy. In the section of a lymph node there appears to be full-fledged malignant tissue in a lymphatic vessel near the node, and I think the pleural nodules are due to malignant tissue in lymphatic vessels. The exact nature of the growth is uncertain; it is unlike an ordinary cancer arising in the bowel and metastasizing in the liver; in certain respects it resembles some primary growths arising from serous membrane, and its structure is not unlike that seen occasionally in reticulum cell sarcoma. I think there is little if any doubt that the essential lesion is a malignant newgrowth, but its exact nature and site of origin are obscure.

[1] Cracium (1931), *Ann. Anat. Path.*, 8: 1089.

DR. PALMER: This case serves to illustrate the all too common difficulty of discovering the diagnosis even when a full post-mortem examination has been made.

DR. ROSE: I think there is one point that the audience would like resolved, and that is the reason for the surprisingly normal blood sedimentation rate. What is the reason, Dr. Johnston?

DR. G. A. W. JOHNSTON: This is sometimes a feature of extensive liver disease. There is, apparently, some disturbance in the manufacture of certain of the plasma proteins concerned in the process of sedimentation.

DR. ROSE: I think we have had a very good discussion today, and each one can go away feeling he has the right diagnosis.

Diagnosis.

A peculiar malignant disease of undetermined type, involving liver, peritoneum, ovaries and pleura, and characterized by unusually severe wasting.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

TESTIMONIAL TO A MEDICAL MAN.[1]

[From the *Australian Medical Gazette*, August 30, 1869.]

ACCORDING to the Argus of the 19th inst the Oddfellows are collecting subscriptions to found a scholarship in the Melbourne University in compliment to Dr. Greeves a past Noble-Grand of that Order to be called the "Greeves M.U. Scholarship" open to the Sons of all Oddfellows. Mr. Greeves is a very old colonist, having been one of the earliest medical men to settle in Victoria then Port Phillip. Dr. Greeves during his long residence in the colony has had extensive experience of almost every phase of public life: he is moreover entitled to the merit of having been one of the earliest—if not the very first—to introduce benefit clubs into the colony. Whatever cause the Oddfellows may have to thank this gentleman, we fear his professional brethren have little reason to feel indebted to one who can lay claim to the very questionable distinction of having first introduced into Victoria the unprofessional and degrading practice of tendering for medical services to benefit clubs. For our part we think it would redound much more to the credit of Oddfellows and others to remunerate their unfortunate, ill paid and hardworked medical officers more in accordance with their services, than to spend large sums of money in empty compliments and frippery.

Justice should have precedence of generosity.

Correspondence.

F.O.C.L.A. POST-GRADUATE WEEK.

SIR: The fifth annual post-graduate week of the Federation of Country Local Associations is to be held at Lismore from August 13 to 17 this year.

Past courses have been held at Armidale, Orange, Canberra and Wollongong. The emphasis is on the work of the country general practitioners, and the course has gained a well-deserved reputation from those enthusiasts who have followed it. With the assistance of the Post-Graduate Committee in Medicine, F.O.C.L.A. plans to make this course an equally outstanding success, and the North Eastern Medical Association extends to all those interested a very sincere invitation to visit the north coast.

These courses have, as their primary objective, the medical education of our members; they also offer an equally important opportunity for meeting one's colleagues and do much good by giving that free exchange of thought in problems which we are all called to face—be they medical, social or medico-political.

Details of the programme and of travel facilities will be published and accommodation will be gladly attended to by the course secretary, Dr. N. Kerkenezov, Lismore.

Yours, etc.,
J. GRIBBEN,
President, Federation of Country Local Associations.

Lismore,
New South Wales,
April 6, 1956.

TOXICITY OF "XYLOCAINE".

SIR: After reading the case history of a death following the injection of 40 cubic centimetres of "Xylocaine" 2% (0·8 gramme—that is, four times the maximum dose or twice the maximum dose if epinephrine is added, as indicated in the manufacturer's leaflet) in THE MEDICAL JOURNAL OF AUSTRALIA, April 7, 1956, I feel it my duty to enthusiasts of local anæsthesia to point out some common mistakes and make some relevant comments so further deaths will not occur:

1. Pregnant women are more susceptible to collapse from local anæsthetics due to the vascular condition of the perineum.

2. The proper quantity of a local anæsthetic to be used is the minimum amount that will produce satisfactory anæsthesia even though you have to wait ten minutes.

3. The anæsthetic was injected much quicker than is compatible with safety. Five cubic centimetres of procaine injected into a vein will give a fit lasting two minutes (Pitkin's "Conduction Anæsthesia").

4. The toxicity depends on the rate of injection and not on the total quantity injected as far as convulsions are concerned.

5. A combination of a local anæsthetic into the perineal body and a short chloroform ether anæsthetic lasting two minutes will be safer, which I have done in 400 obstetric deliveries and 70 Cæsarean sections in the same manner.

I would like to thank Dr. Poidevin for bringing this case to our notice in the journal. Dr. Greenhill, editor of "The Year Book of Obstetrics and Gynecology" in the United States of America, has been an advocate of local anæsthesia for many years.

Yours, etc.,
J. N. R. STEPHEN.

229 Hunter Street,
Newcastle,
New South Wales.
Undated.

CORONARY DISEASE AND MYOCARDIAL INFARCTION.

SIR: I have read with great interest and satisfaction the letter of Dr. F. X. Garvan Hurley on the age-old topic of the relative association of myocardial infarction with coronary "occlusion", the inverted commas advisedly for a reason referred to later.

I must say that after reading the review in "Current Comment" of the article by Snow *et alii*, I was unimpressed as to any added help it provided for those of us more especially concerned with the evaluation of coronary syndromes, and it is in this particular that I wish to comment.

My main itch centres around the aspect of the clinician being relegated in the pathologists' discussion and invited to return to the era prior to 1940 and believe that, after all this time, "coronary occlusion almost inevitably results in infarction" and so, inferentially, the electrocardiogram may, at times, fail to detect it. I thus quote from the article by Snow *et alii*: "It became easy for the pathologist to accept conspicuous coronary occlusion in the absence of demonstrable infarction, and when a prolonged attack of cardiac pain occurred without the associated changes of infarction in the electrocardiogram, the clinician was ready to attribute it to coronary occlusion without infarction." The reference is, of course, to the findings of Blumgart *et alii* (1940).

When, oh when will the "reading public", Dr. Hurley's obvious concern, be instructed in the concept of physiological coronary occlusion? This somewhat coined term may be transitory when initiated by spastic vascular phenomena, or more prolonged, yet reversible, as a result of accidents

[1] From the original in the Mitchell Library, Sydney.

to the *vasa vasorum* when they extend to the intimal layer. The ischæmia resulting from these happenings, especially if prolonged and repeated, would result in morbid changes, ranging from acute necrosis to varying stages of fibrosis. Having in mind these multiples at issue, it is difficult to see how the matter of relating the age, by a week or two, of contiguous areas of healing to the collateral circulation, can be resolved by Mallory's differential stain as indicated by Snow *et alii*.

Further, we would like the pathologist to know that we are not necessarily disturbed in our assessment when faced with the situation of "a prolonged attack of cardiac pain without the associated changes of infarction in the electrocardiogram". In this particular I wish to quote, sir, what I had to say in a previous issue of THE MEDICAL JOURNAL OF AUSTRALIA: "In the eyes of the pathologist there was probably no more mistermed condition in medicine than the coronary episode, and there was much need for a better understanding between the pathologist and the clinician as to the implications underlying its mechanism in atheroma. The clinical impression and laboratory evidence were not necessarily inconsistent with necropsy findings as was commonly supposed. As an illustration, an intimal hæmorrhage might escape detection unless it was meticulously searched for, and yet initiate ischæmia resulting in sudden death from ventricular fibrillation." Further, we are well aware that unless a special technique is used, an intramural infarct may not be revealed electrocardiographically.

Because of a partiality for the belief that the thrombogenic atheroma hypothesis of Duguid and that of the lip of Goffman and company are complementary, I am not quite happy about Dr. Hurley's seeming reluctance to equate the two viewpoints, the more so because of a personal knowledge, in the case of the people named, of a degree of respect for the other's work.

I am in entire accord with Dr. Hurley in his concern for proper guidance in this subject. The fact that you, sir, have so generously extended your columns to him, though the unwitting subject of his impeachment, is proof, if such were necessary, that you are no less concerned than he that the standard of medical education of THE MEDICAL JOURNAL OF AUSTRALIA be maintained. Further, I do not consider that your sub-editor of the review in question has done anything so terribly wrong, and he certainly merits praise in having provoked a very critical exposition from one very informed in this subject. My predilection for blame in this matter centres around those who, though versed in electrocardiographic theory, use terms loosely, and so fail in their duty to instruct the referring physician either for their convenience or his.

Yours, etc.,

Sydney, W. J. MCCRISTAL.
April 6, 1956.

SURGERY OF HAND TRAUMA.

SIR: Mr. Peter Greenwell's excellent address, and the resulting discussion on the treatment of hand injuries, which appeared in THE MEDICAL JOURNAL OF AUSTRALIA of March 31, 1956, is a timely reminder to all hospital authorities and surgeons having to do with these injuries that the best, in fact the only good, results follow early and skilful treatment of these injured hands.

Some hospitals in this city have special hand clinics, yet others admit even minor hand injuries straight to the wards for treatment by an experienced surgeon of senior status. However, for some reason or other, we still repeatedly see the unfortunate who, even though suffering from a severe hand or finger injury such as a compound fracture or even severed tendons, has been attended only by a recently qualified graduate in the casualty department of one or other of the smaller hospitals. Such a patient is not admitted. His hand receives merely a perfunctory examination, perhaps the skin wound is stitched with no anæsthetic, the fashionable antibiotic of the moment is prescribed, and the patient is then referred to his own practitioner. Usually he does not see him until the next day, thus losing valuable hours. Then by the time a consultant has been summoned, and hospitalization arranged, more valuable hours have been lost and infection may have supervened; so that the patient may lose weeks of work before the necessary tendon suture or graft can be safely performed.

Surely such a patient could be admitted immediately to the ward and so treated by the appropriate consultants who are attached to all such hospitals. If there is a local bed shortage, could he not be referred immediately, without unnecessary meddling with the injury, to other and larger hospitals where beds are available? This would result in an enormous saving of wasted man hours, with improvement in the results of hand surgery. These good results are ample justification for the long and tedious time necessarily spent on some of the more difficult reparative surgery of these hands.

Yours, etc.,

141 Macquarie Street, THOMAS F. ROSE.
Sydney,
April 5, 1956.

THE TREATMENT OF ULCERATIVE COLITIS.

SIR: In his letter (M. J. AUSTRALIA, March 17, 1956) concerning the ætiology of ulcerative colitis, Dr. J. W. Graham states that my claim that a neurosis is the basic cause goes beyond the evidence.

The evidence quoted by Dr. Graham can only be the evidence he has found himself, directly or indirectly. He can hardly be in the position to assess the evidence available to me. Knowing the nature of the evidence I have found in these cases, and knowing that the same evidence, though present, has been missed by colleagues concerned in the same cases, I can only conclude that this vital evidence has eluded Dr. Graham. The experiences of Leeuwenhoek are roughly analogous. With his crude microscope he could see and describe structures which were not observable to his contemporaries, whether they used his microscope or not. To them the evidence was inadequate.

Perhaps a closer analogy is the story of a certain physician who, for certain reasons, undertook an appendectomy. A surgical colleague kindly stood by. After an unconscionable period of fumbling and sweating, as he searched interminable loops of small bowel and omentum, the whole abdominal cavity had been explored, and the appendix had not been found. He might have said that an appendix had not been present. The evidence that an appendix was present was inadequate. However, the surgeon took over, incised the peritoneal covering of the posterior abdominal wall and displayed the elusive organ.

A letter is not the best place to unfold the mysteries of neurosis, but the following list of less known aspects of neurosis might help in the better understanding of this widespread condition.

1. The hypothalamus and autonomic nervous system is the anatomical basis of the neuroses, in contrast to the cortex and pyramidal system in many other nervous diseases.

2. "Autonomic neurosis" would be more accurate terminology, and a kinder one.

3. Fear is the basis of the hypothalamus and the subconscious, as opposed to reason as the basis of the cortex and consciousness. This is one reason why reason fails to cure an established neurosis. It also disposes of such archaic ideas and expressions as "imaginitis", "imagining" and "there is really nothing wrong", which are still in daily use.

4. Where the autonomic nervous system is the central mechanism concerned, the peripheral component is the plain muscle fibre of some hollow viscus, be the contained fluid blood, air, bile, urine, chyme or fæces. Occasionally it is some other end organ such as a ducted gland.

5. The disordered function of neurosis is dystonia of the peripheral plain muscle fibre, its synergists and antagonists. This is best considered as spasm (for example, *globus hystericus* or *angina pectoris*), and results in pain, which is as real as any other colic; as real in a spinal neurosis as renal colic.

6. The stimulus to set off this chain of reactions may be fear, frustration or repetitive exhaustion. The process is as uncontrollable as renal or biliary colic.

7. If the condition persists for more than a certain, critical period of time, the pattern will be irreversible. The symptoms will persist, even after the initial stimulus is removed.

8. Pain-relieving drugs, whether they be analgesics, hypnotics or cortisone steroids, can easily expedite the irreversible progress resulting in the various "addictions".

9. No known drug is a cure for the neuroses. Drugs will quickly relieve symptoms, but adaptation quickly occurs and the beneficial effect is lost, unless adequate psychotherapy can be given during this short, critical period.

10. If a neurosis persists for more than a certain period, the resultant ischæmia will cause ulcerative, fibrotic and

other "organic" changes known as psychosomatic disease. Psychosomatic disease equals neurosis *plus* organic complications; for example, gastric neurosis (neurosis) can pass to gastric ulcer (psychosomatic disease).

I would like to point out also that, like any other disease, neurosis is no respecter of persons. It can happen to anybody, regardless of their intelligence quotient, mental stature or social status. It can, and does, affect the doctor as well as the rest of the community. There can be no "holier than thou" attitude by the doctor.

If the prevalence of this pathology is understood, and I think it is the basis of at least 30 relatively common diseases of doubtful ætiology, much time and suffering will be saved. If the mechanism of neurosis is kept in mind, especially the role of the disordered neurogenic control of the peripheral plain muscle fibre, most of the difficulties are resolved, especially an appreciation of the fact that these patients suffer real pain.

Another stumbling block is the failure "to respond to supportive treatment and the ministrations of the psychiatrist". Whereas this failure is used as an argument against the ætiology being neurosis, it is in fact characteristic of neurosis. Obsessional neurosis, cardiac neurosis and spinal neurosis, as most orthopædic specialists will agree, are notorious in their failure to be cured by psychotherapy or other measures. Ulcerative colitis, with its unsavoury reputation for cure, certainly qualifies in this respect for membership of the neuroses and psychosomatic diseases.

Yours, etc.,

185 Macquarie Street,
Sydney,
Undated.

Brian Haynes.

Post-Graduate Work.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

Programme for May, 1956.

Courses for Higher Qualifications.

Microbiology.—A course in microbiology suitable for candidates for the M.D., the M.S. and the diplomas will commence on May 15 at 3.45 p.m. at the Bacteriology Department, University of Melbourne, and continue on Tuesdays at 3.45 p.m. for thirteen to fifteen weeks. The fee for this course is £8 8s., and enrolments should be made with the Post-Graduate Committee on their prescribed form and accompanied by the fee by May 1.

Radiodiagnosis.—A course in radiodiagnosis suitable for D.D.R. and D.C.R.A. candidates will commence on May 7. Lectures will be held at 4.30 p.m. on Mondays and Thursdays till July 23, and where possible will be held in the X-ray departments of hospitals. The fee for the course is £18 18s., payable to the Post-Graduate Committee. Enrolments for single lectures are at the rate of £1 1s. each. Entries, on the committee's forms, will be received till April 23. The committee has arranged this course in consultation with the College of Radiologists of Australasia.

Otology and Laryngology.—A course in otology, laryngology and special pathology for D.L.O. candidates will be conducted by the Ear, Nose and Throat Section of the Victorian Branch of the British Medical Association from May 1 till mid-August, chiefly on Tuesdays and Thursdays, at 5 p.m. Enrolments should be made as soon as possible through the Melbourne Medical Post-Graduate Committee, from whom a timetable will be available.

Psychiatry and Anæsthetics.—For courses in psychiatry and in anæsthetics see below.

Overseas Lecturers.

Professor F. R. Winton, M.D., D.Sc., Dean of the Faculty of Medicine and Professor of Pharmacology at University College, London, will visit Melbourne from May 2 to 7. He will lecture in the Medical Society Hall at 8.15 p.m. on Thursday, May 3, on "Pharmacology of Renal Tubules". The fee for this lecture will be 15s., but those who have paid an annual subscription to the committee are invited to attend without further charge.

Professor David Whitteridge, F.R.S., B.Sc., D.M., Professor of Physiology in the University of Edinburgh, will visit Melbourne from May 10 to 16. He will attend seminars at the University arranged by the Departments of Anatomy and Physiology, to which members of the medical profession are invited. Details may be obtained from the Post-Graduate Committee. His work includes investigations in the cardio-vascular disturbances in paraplegia, the effects of the distension of viscera in man, the nervous control of eye movements and other subjects.

Sir Lionel Whitby, C.V.O., M.C., M.A., M.D., F.R.C.P., Regius Professor of Physic and Master of Downing College in the University of Cambridge, who will visit Melbourne as the Sims Commonwealth Travelling Professor from May 25 to June 8, will give an open lecture to the medical profession on Friday, June 1. This will be on "Anæmia in General Practice" and will be held at 8.15 p.m. in the Medical Society Hall.

Psychiatry.

A course in psychiatry will be conducted by the Victorian Mental Hygiene Authority from April 7 till July 3. This will consist of a series of lecture-demonstrations on Tuesday evenings from 8 p.m. till 10 p.m. There will be two lectures each evening, held in the Mental Health Research Institute, Royal Park Receiving House, Poplar Street. The programme includes a short symposium on psychotherapy. The course is open to all medical practitioners, and there is no fee for attendance. Inquiries should be directed to the Chief Clinical Officer, Mental Hygiene Authority, 300 Queen Street, Melbourne.

Attention is also directed to departmental case conferences held in the metropolitan area, and to bi-monthly meetings of the Mental Hygiene Medical Officers' Association and the regular meetings of the Australasian Association of Psychiatrists. The above course will be followed by a series of twelve lectures to be conducted by the Australasian Association of Psychiatrists commencing on July 10.

Post-Graduate Lectures to be Conducted by the Faculty of Anæsthetists.

All members of the medical profession are invited to attend a series of lectures to be conducted on various dates from April till August. Inquiries should be addressed to the Honorary Secretary, Victorian State Committee of the Faculty, Royal Australasian College of Surgeons, Spring Street, Melbourne.

Country Courses.

Bendigo.—At Bendigo on Friday, May 18, at 8 p.m. at the Base Hospital, Dr. J. A. Game will speak on "Common Neurological Problems". Dr. A. J. Walters, 514 High Street, Golden Square, Bendigo, is the local secretary.

Koroit.—At Koroit on Saturday, May 19, Dr. H. B. Kay will speak on "Diagnosis and Management of Coronary Disease", followed by Dr. Kate Campbell on "Neonatal Diseases". Dr. W. R. Angus, 214 Koroit Street, Warrnambool, is the local secretary for this course.

Fees.—Fees for the above two courses are at the rate of 15s. per lecture, but those who have paid an annual subscription to the committee are invited to attend without further charge.

Flinders Naval Depot.—At Flinders Naval Depot on Wednesday, May 9, at 2.30 p.m., Dr. J. M. Gardiner will discuss a cardiac subject. This is by arrangement with the Royal Australian Navy.

Inquiries.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

F.O.C.L.A. POST-GRADUATE WEEK.

The fifth annual post-graduate week arranged by the Federation of Country Local Associations in New South Wales will be held at Lismore from August 13 to 17, 1956. The programme is as follows:

Monday, August 13: 9 a.m., registration; 10 a.m., official opening (Right Honourable Sir Earle Page); 11.30 a.m., Memorial Oration, "The Late Dr. E. Woodward", Dr. R. E. Longworth; 2 p.m., "Gastric Ulcer: Some Interesting Aspects", Dr. T. Greenaway; 3.45 p.m., "Indications for Gastrectomy", Dr. Evan Thomson.

Tuesday, August 14: 9.30 a.m., "After Gastrectomy", Dr. Evan Thomson; 11 a.m., "Disordered Uterine Action",

Professor Shedden Adam; 2 p.m., "Hypertension Symposium", Dr. T. Greenaway (medical), Professor Shedden Adam (obstetrics), Dr. E. Thomson (surgical); afternoon tea; 3.45 p.m., "Some Hazards of Multiparity", Professor Shedden Adam; 8 p.m., open lecture.

Wednesday, August 15: 9.30 a.m., "Some Childhood Maladies in General Practice", Dr. H. Stuart Patterson (College of General Practitioners—Queensland Faculty); morning tea; 11 a.m., "Emergencies in General Practice", Dr. B. Cook, Dr. R. J. Jackson, Dr. Millar, Dr. A. Rumore (College of General Practitioners, New South Wales Faculty).

Thursday, August 16: 9.15 a.m., "Jaundice in Childhood", Dr. Felix Arden; 10.15 a.m., "A Recent Review of American Medicine", Sir Alexander Murphy; 11.30 a.m., "Psychosomatic Diseases in Children", Dr. Felix Arden; 2 p.m., "Rheumatic Fever Symposium", Dr. Felix Arden, Sir Alexander Murphy, Dr. T. Greenaway; 4.15 p.m., "Obesity", Professor J. H. Sheldon (overseas lecturer).

Friday, August 17: 9.30 a.m., "Anorexia", Professor Sheldon (overseas lecturer); 11 a.m., "The Prevention and Treatment of Cardiac Invalidism", Sir Alexander Murphy; 2 p.m., clinico-pathological discussion, Dr. T. Greenaway (chairman); 3.45 p.m., meeting of delegates, medico-political meeting.

Public Health.

POLICE OFFENCES (AMENDMENT) ACT, 1908, AS AMENDED, OF NEW SOUTH WALES.

THE Under Secretary, Chief Secretary's Department of New South Wales, has requested that publicity be given to the following proclamation, which it is proposed to gazette on Friday, June 8, 1956, applying Part VI of the *Police Offences (Amendment) Act* to α-acetylmethadol and certain other drugs:

PROCLAMATION.

J. Northcott,

Governor.

I, Sir John Northcott, Knight Commander of the Most Distinguished Order of Saint Michael and Saint George, Knight Commander of the Royal Victorian Order, Companion of the Most Honourable Order of the Bath, Lieutenant-General on the Retired List of the Australian Military Forces, Governor of the State of New South Wales and its Dependencies in the Commonwealth of Australia, with the advice of the Executive Council, do, by this my Proclamation, declare that Part VI of the Police Offences (Amendment) Act, 1908, as amended, shall apply to:

α-Acetylmethadol (α-6-dimethylamino-4,4-diphenyl-3-acetoxyheptane), its salts and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of α-acetylmethadol.

α-Methadol (α-6-dimethylamino-4,4-diphenyl-3-heptanol), its salts and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of α-Methadol.

β-Acetylmethadol (β-6-dimethylamino-4,4-diphenyl-3-acetoxyheptane), its salts, and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of β-acetylmethadol.

Dimethylthiambutene (3-dimethylamino-1,1-di-(2-thienyl)-1-butene), its salts, and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of dimethylthiambutene.

Ethylmethylthiambutene (3-ethylmethylamino-1,1-di-(2-thienyl)-1-butene), its salts and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of ethylmethylthiambutene.

6-methyl-Δ^6-desoxymorphine, its salts and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of 6-methyl-Δ^6-desoxymorphine.

Isopropyl and other esters of 1-methyl-4-phenylpiperidine-4-carboxylic acid, their salts, and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of isopropyl or other esters of 1-methyl-4-phenylpiperidine-4-carboxylic acid.

6-Dimethylamino-4,4-diphenyl-3-hexanone, its salts and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of 6-dimethylamino-4,4-diphenyl-3-hexanone.

β-Methadol (β-6-dimethylamino-4,4-diphenyl-3-heptanol), its salts and any preparation, admixture, extract or other substance containing not less than one per centum of β-Methadol.

Dihydrohydroxymorphinone, its salts and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of dihydrohydroxymorphinone.

Signed and sealed at Sydney this fourteenth day of March, one thousand nine hundred and fifty-six.

By His Excellency's Command.

C. A. KELLY.

GOD SAVE THE QUEEN!

Obituary.

EULALIA RICHARDS.

THE late Dr. Eulalia Richards was born at Battle Creek, Michigan, United States of America, on August 22, 1878, and died peacefully at Manly, Sydney, on March 15, 1956.

Training as a physician in her home State, she was registered M.D. in 1902, after which she visited Britain for further study and gained the diplomas of L.R.C.P. and S. (Edin.) and L.R.F.P.S. (Glasgow). Hospital work, travelling and lecturing kept her there until 1906, when she sailed to Australia and resided mainly in Sydney until her death.

From 1906 to 1913 she was on the staff of the Sydney Sanitarium and Hospital, Wahroonga, as physician, anæsthetist, and lecturer to trainee nurses. In 1912 she published "Ladies' Handbook", a guide to women in the general principles of home-making, child welfare and healthful living. From 1913 to 1926 she was engaged in suburban and Macquarie Street private practice, giving liberally of her time and honorary services to various women's hospitals, including the Crown Street Women's Hospital and the Rachel Forster Hospital; and also to the Saint John Ambulance Association, of which order she was made an "associate serving sister".

In 1926 she returned to the staff of the Sydney Sanitarium and Hospital where she spent nearly all of her time until her retirement. In 1932 she was given leave of absence to do post-graduate study in London in obstetrics, and to visit her people in the United States of America.

She gave over thirty years to her hospital appointment, serving the sick and as teacher and lecturer. In the training of nurses, she always strove to uphold the highest standards of efficiency and devotion to duty. Her ministry to the sick always had as its aim, not only restoration to physical health, but also strengthening of mental and spiritual defences.

In her home life she was a devoted mother to her three children, and it was in her home and garden that her interests were centred on her retirement some nine years ago after a very full life devoted to what she considered more than a mission—in fact, her inspiration and the very reason for living.

JOHN JOSEPH WITTON FLYNN.

DR. J. D. RUSSELL writes: Over many years I had the privilege of friendship with John Flynn. Returning recently from overseas, I was shocked to hear of his death. John was such an alert vivid outgoing man with a zest for life and affairs that it is hard to realize that he has passed from the contemporary scene. In these days, when society places great value on conformation and distrusts any deviation from the mean of character and temperament, rare spirits like John Flynn are a joy to behold. He was ever a sturdy individualist, a forthright man and a doughty fighter for that which he thought was right, deeming tact not always to be a virtue. He demanded a high standard of ethical

behaviour, yet had little time for pomposity or pretence. He enjoyed the give and take of controversy and was at his best at the verbal level. Let it be said that he was mostly on the giving end, as few could match him in ready wit and repartee. Yet withal, not many would take offence, and no doctor in Sydney had so many friends nor knew so many people in all walks of life. His interests were manifold—professional, sporting, social and the services.

But all this was the façade, the *persona*, the mask. Underneath John Flynn had his reserves. The loss of an eye in France in the 1914-1918 war was a considerable and, at times, a painful handicap. Of late years, too, his health was materially impaired. He had his warnings, but chose typically to ignore them. The words of the one-time Dean of Saint Paul's come to mind: "Never send to know for whom the bell tolls; it tolls for thee." John Flynn had heard the bell tolling, yet never complained, never discussed his health and was unprepared to restrict his many activities; and only two years ago he undertook the responsible position of President of the New South Wales Medical Board. His reserve extended to all his deeper feelings—for his family, his religion and his love of country. I know that his ancient faith meant much to him and was ever a material influence on his life and attitudes. Air Vice-Marshal E. A. Daley has ably sketched already his distinguished service in two world wars, and his peace-time interest in the Royal Australian Air Force and ex-servicemen through the Repatriation Department.

And so it is that John Flynn was an uncommon man, possessing all his days the rare qualities of courage and honour—virtues I believe that eagleplume men's souls and set them apart.

I like to think and verily believe that he died as he would wish—in the very midst of living, at the end of a long day of endeavour, in his home, of which he was so justly proud, in the comforting presence of his wife and daughter, whom he loved so well.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

The following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act*, 1938-1955:

Registered medical practitioners who are required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) of the Act (the qualification being, unless otherwise stated, M.B., B.S., 1956 (Univ. Sydney)): Alexander, David Adolph; Allen, Bruce Mitchell; Allen, Peter Barclay; Allison, George Harris Cory; Andrews, Bruce Allan; Ashley, Bruce Charles Earnfaunce; Augulis, Visvaldis; Bailey, Richard John; Barker, Lynette; Barratt, John Gordon; Barry, Grahame Maxwell; Bashir, Marie Roslyn; Bassett, Duncan James; Bell, Sydney Malcolm; Benjamin, Bruce Neil Proctor; Blazejowski, Bronislaw Alexander; Bosanquet, Phillip Walter George; Brien, William Robert; Buhagier, Anthony Roland; Burke, Claire Agnes; Burke, Denis Timothy; Burman, Ian Ronald; Cabrera, Kenneth Francis; Carew, Bernard John; Carroll, Anthony; Carter, Jeanette Jill; Cartmill, Timothy Boyd; Castle, Marian; Challis, Shirley Shebah; Child, Donald Stewart; Claremont, Peter Francis; Coffey, Rosemary McVeigh; Cookson, Philippa Nancy; Coombes, Bevan Harvey; Cowle, Joffre Bartholomew; Cridland, Marion Diana; Cronin, Anthony Earl; Cummings, Royal; Daly, Ian Charles; Daniel, Bruce Ramsay Crofton; Davis, Alan Edward; Davis, John Raymond; Davis, Penelope Jan; Degotardi, Peter Richard; Domany, Susan; Drew, John Harvey; Dwyer, Francis Norman; Edwards, Peter Drummond; Ellis, Max Harvey; Farlow, John Lyle; Fay, Warwick Harvey; Fowler, Francis Bursill; Fraser, Thomas Aird; Fry, Hunter John Hall; Fuller, Kevin Maxwell; Gale, William Colin; Gentile, John William Julius; Gibson, Geoffrey Reginald; Goddard, Kenneth Ernest; Grainger, Peter Francis; Greening, Dorothy Grace; Hardacre, Leslie Brian; Harrison, Frank George; Harrison, John Anthony; Hart, Graham Hugh Basil; Hepburn, Susan Frances; Herron, John Tennent; Hinde, Frederick Charles; Holt, Raymond Stuart, M.B., B.S., 1956 (Univ. Sydney), B.Sc., Med., 1954 (Univ. Sydney); Hosking, Alan Thomas; Howe, Geoffrey David; Hughes, Peter Robert; Humphry, Paul Stanley; Inall, John Albert; Jackson, Jacqueline Ann; Jenkins, Elaine Linda; Johnson, James Ronald; Kennedy, Michael Andrew; Kernot, Laurence William; Khoo, Anthony Keng Beng; Ko, Florence Hui Liau; Lai, Min Tet; Lambert, Leon Warren; Lang, John Keith; Lang, Marie Elizabeth; Langford, Mae Vimiera Adelaide; Leber, Carl Frederick; Lee, Martin Robert; Lloyd, Elizabeth Vera; Logan, Geoffrey Gibson; Loughman, James; Lovric, Vladimir Albert; Lucey, Maureen; McClure, Eunice Mary; McDonell, John Alexander; McCredie, Richard Michael; Mackie, Ian John; Malcolm, Susanne Foster; Manuel, Leela Therese; Marks, Harry; Miller, Jacques Francis Albert Pierre; Mistilis, Steven Paul; Monaghan, William; Morgan, Bruce Alexander; Morgan, Gerald John; Muggridge, William Alexander; Newman, John Warwick; Nichols, James Johnstone; Nott, David Bruce; Novotny, John; Oakeshott, Robert John; Orr, Colin Montague; Owen, Michael Samuel; Oystragh, Philip; Parnell, Peter Montague; Parry, Eric Leonard; Pascoe, Barry Joseph; Penney, Douglas James; Pittar, Graham; Pittar, Yorke Arthur; Playoust, Marc Roger; Potts, John Gilroy; Pryde, Ian Noel; Purchas, James Maurice; Ramrakha, Ramchandra; Ramrakha, Shiu Chandra; Reading, Bruce David; Reid, Leslie Robert; Renouf, Noel Arnold; Roberts, Lindsay Milton; Roche, James Barry; Roebuck, David John; Rogers, Shirley Isobel; Rose, Gabriel; Rothwell, Frederick Leigh; Russell, Damaris Kaye; Sarks, John Peter; Schutta, Henryk Szczesnt; Siddall, David James; Sloane, David Rae; Smartt, Herbert Henry; Smidlin, Richard John; Smythe, Allen Douglas; Speechley, Ronald Alwyn; Stack, Ellen Mary; Standish, Patricia Anne; Starr, Gwenneth Nancy; Stylis, Stanley Chris; Sundstrup, Bertel; Sutherland, Donald Fyfe; Swanton, Alastair William; Tarlinton, Michael John Kevin; Thorburn, Geoffrey Donald; Tinsley, Richard William; Toakley, Jean Larrey; Tottenham, Ronald Charles; Tyer, Harry Danvers David; Vines, Arthur Peter; Wald, Louis; Walder, Brien Keith; Walshe, Annette Maria; Walshe, Isobel Margaret Ann; Warner, Eva Ruth; Wayland, Jill Pamela; Weinrauch, Lindsay Elizabeth; Wonson, Ronald Charles; Wren, Barry George; Yeo, John Douglas.

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*a*) of the Act:

Alichin, Eric Edward, M.B., B.S., 1953 (Univ. Melbourne); Atlas, Zygfryd, M.B., B.S., 1954 (Univ. Melbourne); Cossins, Graham Alan, M.B., B.S., 1953 (Univ. Queensland); Jolley, John Bedlington, M.B., B.S., 1953 (Melbourne); Lustig, Fred Walter, M.B., B.S., 1954 (Univ. Melbourne); Maguire, Maurice Glen, M.B., B.S., 1953 (Univ. Adelaide); Ricketts, Graham, M.B., B.S., 1949 (Univ. Queensland); Thompson, John Evlyn, M.B., B.S., 1955 (Univ. Adelaide); Webling, Donald D'Arcy, M.B., B.S., 1955 (Univ. Adelaide); Gray, Ronald Wallace Menzies, M.B., B.S., 1944 (Univ. Sydney).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*b*):

Bell, Alan, M.B., B.S., 1941 (Univ. Durham); Cantwell, John Thomas, M.B., B.Ch., 1946 (National Univ., Ireland), L.M., 1948 (Ireland); Barnard, George Edward, L.M.S.S.A. (London), 1942, L.R.C.P. (Edinburgh), 1942, L.R.C.S. (Edinburgh), 1942, L.R.F.P.S. (Glasgow), 1942.

The following additional qualifications have been registered: Brown, Judith Macarthur, D.O. (London), 1953; Champion, Maret, D.C.H. (London), 1955; Gates, Bruce Forsyth, D.A., R.C.S. and R.C.P. (England), 1955; Ham, Harold John, D.M.R.E. (Cambridge), 1933, F.F.R. (London), 1939, F.C.R.A., 1949; Jeffrey, George Pearson, D.Obst., R.C.O.G. (England), 1955; Rigg, Charles Andrew, M.R.A.C.P., 1955; McGovern, Vincent John, M.D., 1953 (Univ. New Zealand), F.R.A.C.P., 1954; Morris, Walter John, F.R.C.S. (England), 1954, F.R.C.S. (Edinburgh), 1954; Tully, John Kitching, D.Obst., R.C.O.G., 1951, M.R.C.O.G. (London), 1955.

The Royal Australasian College of Physicians.

ANNUAL MEETING.

At the annual meeting of The Royal Australasian College of Physicians held in Wellington, New Zealand, in March, 1956, the following office-bearers took office for the period 1956-1958:

President: Dr. E. G. Sayers (New Zealand).

Vice-Presidents: Dr. Bruce Hunt, M.B.E. (Western Australia), Dr. Clive H. Fitts (Victoria), Dr. C. R. Burns, O.B.E. (New Zealand).

Censor-in-Chief: Dr. T. M. Greenaway (New South Wales).

Honorary Treasurer: Dr. W. P. MacCallum, C.B.E., D.S.O., M.C., E.D. (New South Wales).

Honorary Secretary: Dr. H. Maynard Rennie (New South Wales).

Past President: Dr. C. G. McDonald, C.B.E. (New South Wales).

At this meeting, with Dr. E. G. Sayers, the President, in the chair, the following were elected to Fellowship: Dr. A. J. Barnett, Dr. J. J. Billings, Dr. H. W. Garlick, Dr. D. R. Gauld, Dr. E. A. North and Dr. R. F. A. Strang, of Victoria; Dr. S. P. Bellmaine, Dr. A. E. McGuinness and Dr. R. B. Perkins, of New South Wales; Dr. O. Hirschfeld, of Queensland; Dr. E. G. Saint, of Western Australia; Dr. E. B. Sims and Dr. R. T. Binns, of South Australia.

The following, having satisfied the Board of Censors in New Zealand, were admitted to membership by the President: Dr. J. Apthorp, of Hutt Valley; Dr. D. W. Beaven, of Christchurch; Dr. M. I. Bostock, of Hastings; Dr. D. S. P. Dickson, Dr. G. L. Glasgow and Dr. J. D. Sinclair, of Auckland; Dr. P. Fleischl and Dr. J. V. Hodge, of Dunedin; Dr. M. J. A. McDonald, of Rotorua; Dr. A. R. Stone, of Wanganui; Dr. V. I. E. Whitehead and Dr. A. C. H. Wilkinson, of Lower Hutt.

Dr. R. H. Black and Dr. H. M. Whyte, of New South Wales, were admitted to membership under the special provisions of Article 37.

Naval, Military and Air Force.

HONOURS AND AWARDS.

THE following honour is made in connexion with the Australian Military Forces (*Commonwealth of Australia Gazette*, Number 12, of March 15, 1956).

THE AUSTRALIAN EFFICIENCY DECORATION.

4/31901 Colonel Richard Geoffrey Champion de Crespigny, Royal Australian Army Medical Corps.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 12, of March 15, 1956.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointments.—Surgeon Commander George Clifford Denny is appointed on loan from the Royal Navy, with seniority in rank of 30th June, 1951, dated 9th December, 1955. Joseph Raymond Clark is appointed Surgeon Lieutenant (for Short Service) (on probation), dated 19th January, 1956.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointment.—Peter Hardy Smith is appointed Surgeon Lieutenant, dated 29th November, 1955.

Promotion.—Surgeon Lieutenant John Henry Begg is promoted to the rank of Surgeon Lieutenant-Commander, dated 15th January, 1956.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

1/8062 Captain J. K. O'Reilly relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command), 17th January, 1956.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/59168 Major (Honorary Lieutenant-Colonel) R. K. Wilson is placed

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 7, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	5(3)	4(3)	3(1)	..	..	..	..	..	12
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	2	..	20(20)	..	..	..	..	..	22
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3(1)	16(14)	5(5)	..	7(7)	..	..	..	31
Diphtheria	..	6(6)	1	..	3(2)	..	..	..	10
Dysentery (Bacillary)	..	4(4)	5(2)	..	4(3)	..	..	..	13
Encephalitis	3	4	..	..	1	..	..	..	8
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1(1)	..	..	..	..	..	..	1
Infective Hepatitis	100(37)	124(38)	..	10(6)	7(3)	..	1	4	246
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	4(1)	..	..	..	..	..	4
Malaria	..	..	1(1)	..	..	..	..	..	1
Meningococcal Infection	1	5(5)	..	..	..	..	..	..	6
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	11(7)	12(10)	3(3)	7	6(2)	1	..	..	40
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	28(24)	..	4(2)	2(2)	..	..	..	34
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	18(6)	30(15)	2(1)	1(1)	1	..	..	..	52
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	3	..	..	..	3
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	18(16)	9(6)	5(1)	7(6)	11(7)	3(1)	..	..	53
Typhoid Fever	..	..	1	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	3(2)	..	..	..	..	..	3
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

upon the Retired List (Northern Command) with permission to retain his rank and wear the prescribed uniform, 27th January, 1956. To be Majors: 1/10316 Captain (Temporary Major) H. W. A. Forbes, 26th January, 1956, and 1/28390 Captain I. H. Chenoweth, 27th January, 1956. To be Temporary Major, 27th January, 1956: 1/61808 Captain R. H. Orr.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/121998 Captain (provisionally) J. Beveridge relinquishes the provisional rank of Captain, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), and is granted the honorary rank of Captain, 4th December, 1955.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 3/114023 Major C. A. Jones is confirmed.

Central Command.

Royal Australian Army Medical Corps (Medical).—The provisional ranks of the following officers are confirmed: Captains 4/32002 A. R. Anderson and 4/32035 R. Britten-Jones.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

Northern Command.—To be Honorary Captains: James Burston Webster, 30th December, 1955, and Paul Barte and Reginald Hastings Williams, 3rd January, 1956.

Southern Command.—To be Honorary Captain, 2nd January, 1956: William James Granger.

Notice.

POST-GRADUATE GOLF MATCH.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the Stableford competition for the Post-Graduate Golf Cup and the Brydon Golf Cup will take place on Friday, May 18, 1956, at the Royal Sydney Golf Club, Rose Bay. The Brydon Golf Cup is open to country members attending the annual general revision course, and the following are eligible to compete in the Post-Graduate Golf Cup: (*a*) post-graduate students attending the general revision course, 1956, and those who have attended any courses conducted by the committee during the previous twelve months; (*b*) visiting lecturers; (*c*) members of the Post-Graduate Committee in Medicine and its subcommittees; (*d*) honorary medical officers and resident medical officers of hospitals participating in post-graduate work in New South Wales; (*e*) teachers in the Faculty of Medicine of the University of Sydney, including members of the honorary medical staffs of all clinical schools; and (*f*) members of the annual subscription course.

Applications to compete should be made on the prescribed form, copies of which are available from the committee, and should be lodged with the Course Secretary, 131 Macquarie Street, Sydney, as soon as possible, but not later than Friday, May 4, 1956. The entry fee is £1 (including luncheon) or 10s. (without luncheon), and must accompany the application.

SPECIAL GROUP ON AVIATION MEDICINE (BRITISH MEDICAL ASSOCIATION).

THE Special Group on Aviation Medicine (British Medical Association) will hold a scientific meeting on Wednesday, May 16, 1956, at 8 p.m. at the Physiology Department, Third Floor, New Medical School, University of Adelaide, Adelaide. At this meeting the aero-medical laboratory will be open for inspection, and the officer-in-charge will outline the work carried out in the laboratory and demonstrate the equipment used. In addition, a film entitled "The Partial Pressure Suit" will be shown.

All members of the British Medical Association interested in aviation medicine are cordially invited to attend the meeting.

Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Coombes, Bevan Harvey, M.B., B.S., 1956 (Univ. Sydney); Inall, John Albert, M.B., B.S., 1956 (Univ. Sydney); Miller, Jacques Francis Albert Pierre, M.B., B.S., 1956 (Univ. Sydney); Smythe, Allen Douglas, M.B., B.S., 1956 (Univ. Sydney); Cole, David Ian, M.B., B.S., 1955 (Univ. Sydney); Ferguson, Peter Langbene, M.B., B.S., 1955 (Univ. Sydney); Thomas, Edna Rae, M.B., B.S., 1955 (Univ. Sydney); Asprey, Raymond Vincent, M.B., B.S., 1954 (Univ. Sydney); Farrell, John Caldwell, M.B., B.S., 1954 (Univ. Sydney); Golomb, Minna, M.B., B.S., 1954 (Univ. Sydney); Hathaway, Anthony Clive, M.B., B.S., 1953 (Univ. Sydney); McGovern, Gregory Joseph, M.B., 1953 (Univ. Sydney); Bernard, Henryk Bierzynski, M.B., B.S., 1953 (Univ. Sydney); England, Theodora Margaret, M.B., 1925 (Univ. Sydney), Ch.M., 1954 (Univ. Sydney), D.P.M. (Sydney), 1938; Holmes, Vera, M.B., B.S., 1943 (Univ. Durham); White, Geoffrey Roy, M.B., B.S., 1953 (Univ. Sydney).

Diary for the Month.

MAY 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAY 2.—Victorian Branch, B.M.A.: Clinical Meeting.
MAY 2.—Western Australian Branch, B.M.A.: Branch Council.
MAY 4.—Queensland Branch, B.M.A.: Council Meeting.
MAY 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.